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Dr. Gillette Hayden
and
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for their pioneer efforts and
selfless devotion in behalf
of periodontia and the
American Academy
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Thirty-sixth Annual Meeting

THE AMERICAN ACADEMY OF PERIODONTOLOGY

September 4, 5, and 6, 1952

St. Louis, Mo.

Details of Program on Page 189

DR. HAROLD RAY

Program Chairman

American Academy of Periodontology 1952

Dr. Harold Ray was graduated from Northwestern University, College of Dentistry in 1926 and remained on the staff as a teacher in dental pathology until 1933. He conducted a general practice with emphasis in periodontology in Chicago from 1927 until 1949 and was a graduate student at the University of Illinois from 1946 until 1949 and graduated with the Master of Science degree. He is now associate professor of periodontology at the University of California, College of Dentistry and chairman of the Division of Periodontology, and is practicing periodontology in Palo Alto, California two days per week.

Dr. Ray has written several articles on periodontology and his primary interest is to correlate the histopathology of periodontal disease with its successful therapy. He has been a member of the Academy of Periodontology since 1939 and a member of the Executive Council since 1948 and also a diplomate of the American Board of Periodontology since 1943.

THE JOURNAL OF PERIODONTOLOGY

JULY, NINETEEN HUNDRED FIFTY-TWO

Principles of Correction of Simple Malocclusion in the Treatment of Periodontal Disease*

by I. W. SCOPP, B.S., D.D.S.,* *and* SAUL M. BIEN, B.S., D.D.S., *New York, N. Y.*

THE knowledge and use of orthodontic techniques and skills can contribute materially to the effectiveness of periodontal therapy. Teeth situated in certain unfavorable positions where there is no function or where there is overfunction are predisposed to a diseased periodontium. The physiologic mission of the teeth in relation to supporting structures can best be accomplished in a wholesome biologic relationship to one another and to the periodontal tissues.

Almost always, etiological factors in periodontal disease in a patient are multiple. If malposed teeth cause either underfunction or overfunction or are so situated that the periodontal tissues are being injured during mastication, such teeth should be correctly aligned. The basic tenet of periodontal therapy is that all causative factors in periodontal disease should be eliminated.

It is not difficult to move teeth per se. As long as teeth are in the mouth of a healthy patient, they can be moved. The physiology involved in the movement of teeth is similar to the physiology in the healing of a fractured bone.

In cases where simple appliances will meet the needs of the patient, the periodontist can start treatment with orthodontic consultation, provided he has had some experience and understands the physiology and histo-pathology involved. In difficult cases, treatment as well as consultation by an orthodontist should be sought.

Not until curettage has eliminated all local irritants and all carious teeth have been filled should movement of teeth be undertaken.

HISTORICAL BACKGROUND

As far back as 1766, Woofendale advised that "if the edge of the tooth stands out of its proper direction, it may oftentimes be brought back, provided likewise that the projection of the tooth out of its required direction be not very considerable, and that the pressure does not fall solely on the two neighboring teeth; for it always should be divided by throwing a few turns of the wire or

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ligature over some of those that stand at a distance." . . . He goes on further to discuss occlusal equilibration. "There are many instances in which it is advisable and safe, as well as ornamental to file the teeth; but as it is usually practiced, nothing can be more pernicious. . . . Where a tooth projects beyond the common level and hinders the rest from meeting equally, or receives, itself alone, all the pressure which should fall divided on a whole set, there, filing is necessary. . . ." This is also the best opinion today, shared by both orthodontist and periodontist.

Maury in 1828 advocated the use of ligatures in his Traite Completes. However, he did not make the common contemporary error of using silk or wire ligatures without anchoring them to small metal hooks over the teeth to prevent injury to the soft tissues.

Kneisel in 1836 described the movement of single teeth by means of a gold crown with attached gold springs in *Der Schiefstand der Zahne*, a technique which is still suitable for treatment of periodontal patients today.

Comprehensive basic principles have been amassed in the literature gradually during the past hundred years. It might be well to examine the following axioms propounded by Simon H. Guilford in 1889 in textbook *Orthodontia*, which it would do well to observe: "The resistance at the point from which we exert pressure must be greater than the resistance to be overcome by the pressure. . . . Before applying force it should be seen to that there is space sufficient to accommodate the tooth in the new position it is to occupy. . . ."

It is advisable for the periodontist or general practitioner to confine himself to moving single teeth or small numbers of teeth where this is indicated. A satisfactory orthodontic device can be improvised from a partial denture, bridges, crowns, or even inlays, as described basically in these historical techniques.

In the treatment of cross-bite of one or more teeth for instance, we find that a description by William Robertson in 1835 in his work *A Practical Treatise on the Diseases of the Teeth* devised an appliance for jumping one or two teeth based on the reverse inclined plane as follows: "A gold plate is accurately fitted and firmly secured to the lower range of teeth; it inclines inwardly across the cutting edges of the teeth which inclose the upper incisor. In shutting the mouth, the posterior surface of the irregular tooth acts against the inclined surface of the plate, and by the pressure thus produced, the tooth is gradually moved forward into the proper position. The plate at both ends encloses and rests against the grinding surfaces of the irregular tooth, and prevents the lower edge of the plate from being forced against the gums." Substituting acrylic for gold, we have a modern appliance.

OCCLUSAL FACTORS CAUSING PERIODONTAL DISEASE

The etiologic factors causing occlusal disharmonies are summarized by Leonard:

1. Loss or absence of teeth or contacts resulting in shifting of other teeth and failure in mesiodistal support.

2. Uneven wear resulting in unworn cusps which interfere.

3. Orthodontic defects in which teeth are erupted in unstable position or are so placed as to interfere with free mandible movements.

4. Injurious mouth habits such as habitual clenching of teeth, pushing against teeth with tongue, walking with heavy pipe in mouth, thread biting and other occupational pressures.

5. Periodontal swellings due to inflammation or tumors that push the teeth into position of occlusal interference.

6. Improperly made fillings, bridges and dentures which create occlusal interference or, through faulty clasps, cause lateral thrusts.

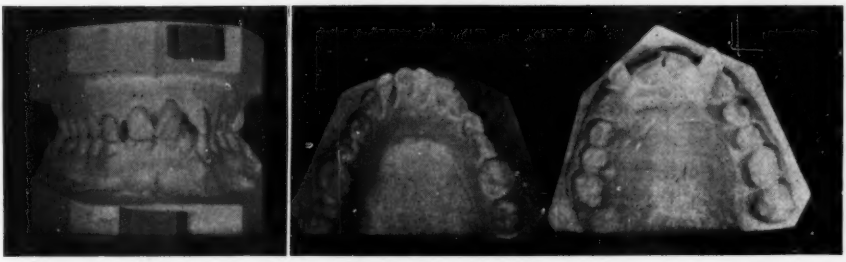

FIGURE 1 — Case I

Before treatment of a 38-year old female. Upper anteriors flaired labially. Spaces between teeth caused food retention. Lack of occlusion of anterior teeth gave rise to a weakened periodontium in this area. There was disuse atrophy of the alveolar bone, periodontal membrane and gingivae. A class II mobility of the upper laterals existed.

7. (Granger) Maximum intercuspation does not coincide with the true centric position. There are strains and stresses.

HISTOPATHOLOGY

Alveolar Bone—Sandstedt and later Oppenheim described the movement of teeth biologically. Wherever continuous pressure is exerted on a periodontium, new bone is formed by osteoblasts. In addition, there is a general rearrangement of the alveolar bone around teeth that are being moved. The even bone plate transforms into spicules of bone arranged parallel to the direction of force. These spicules show resorption on one end and new formation of bone on the other. The tearing down and building up in which the osteoclasts and osteoblasts are prominent continues as long as the orthodontic force is applied. When the force is removed and the teeth are stabilized in the desired position, the bone gradually changes until the solid bone plate is again restored.

Alveolar bone is both a tissue and an organ. As a tissue, it is not static. Throughout life, there is a constant change in the pattern of trabeculae. Bone is resorbed and laid down simultaneously. In disuse, as in certain malocclusions where teeth do not meet their opponents in any movement of the jaw, the supporting cancellous bone of the alveolar process undergoes atrophy. The periodontal fibers become thinner, losing most of their principal functioning fibers. The cementum compensates by becoming thicker.

In movement of teeth or in traumatic occlusion, bone resorption occurs on the side of the pressure and bone is built up on the side of tension. With the fulcrum point of the tooth described by Johnson, Appleton and Rittershofer being somewhere between the apex and the middle of the tooth root, resorption of bone and the building of bone occurs adjacent to the entire surface of the root but on different sides. Areas of resorption are found on opposite sides at the cervical and apical areas of the root. Thus, when a tooth is moved mesially by tilting, the mesial wall undergoes resorption at the crest level of the bone. Apically, the opposite side shows resorption. The movement at the apex is necessarily less than the coronal portion of the root. There may develop a marked increase in the thickness of the periodontal membrane, causing mobility of the tooth. After stabilization of the tooth in a new physiologic position and the physiology of the bone has slowed down and returned to normal, the tooth will be maintained firmly under normal physiological stress.

Very light pressure is needed to move teeth. Excess pressure causes crushing and degeneration of periodontal membrane.

Oppenheim has shown that delicate light lateral stress will show hardly any displacement of the root apex in the opposite direction. The periodontal membrane fibers react to tipping action. About one half of the group of periodontal membrane fibers will be stretched and the other half relaxed, with areas of tension and relaxation diagonally opposite each other.

PERIODONTAL MEMBRANE

A word about the histology of the periodontal membrane is timely. It must be remembered that the osteoblasts and osteoclasts are part of the periodontal membrane. When an excessive force is applied, the periodontal membrane is compressed and may eventually become necrotic. These areas of tissue necrosis have actually been observed by investigators in animal experimentation. If the periodontal membrane is damaged in this way, the function of the osteoclasts and osteoblasts cannot be carried out. This coincides with the clinical observation that intense force, when used in movement of teeth, causes a standstill in the movement of teeth rather than rapid movement. Before teeth are to be moved, a periodontal membrane must be in good health functionally and anatomically.

Kronfeld summarizes the action of force on a tooth as follows: "Powerful tipping forces will cause the apex to move opposite to the direction of the crown, while gentle tipping forces will have so little effect that excursions of the apex may be disregarded. Hence, with delicate force, as is applied in modern orthodontia, the fulcrum is located near the apex; with strong force, the fulcrum is located between the middle and the apical third of the root."

ELONGATION AND DEPRESSION OF A TOOTH

To obtain a harmonious and efficient relationship between the upper and lower jaws, often it is necessary to elongate or depress a group of teeth. Histologically, when a tooth is being elongated, there is a formation of new bone at the fundus of the alveolus. Here the bone spicules are arranged in the direction of pull, parallel to the long axis of the tooth. Again, on the tooth side of the spicules, osteoclasts predominate. Formation of new bone occurs in this way to fill the space that would be created as the tooth is elongated.

Teeth may be depressed in the socket. The bone changes are the opposite to elongation. There is resorption of bone on the inner surface of the socket and in particular on the fundus of the alveolus. This resorption of bone permits intrusion of the teeth.

PRINCIPLES OF MOVING TEETH

Certain principles of moving teeth should be kept in mind. These are listed below:

1. Sufficient space should be made available for the tooth. This should be measured accurately using either calipers or study models, the teeth of which have been cut off and replaced on wax in the desired position.

2. Forces should be distributed so that the teeth which are not to be moved remain in proper position.

3. The concentrated force should move the teeth in the direction required.

4. After the teeth are moved, they must be retained in position with splints which are either fixed or removable, preferably the former.

5. Pressure must be kept within physiological limits.

6. The teeth should not be damaged by improper leverage.

7. The periodontium should not be injured by poorly designed appliances.

The use of bands and standard orthodontic appliances in inexperienced and untrained hands should not be encouraged. Bien has shown that an apparently light primary force can, by compound leverage

on an ordinary appliance, exert excessive displacement forces unless controlled under competent orthodontic supervision. Orthodontic appliances are far too dangerous to the teeth, mouth and the reputations of all involved unless used with the greatest degree of skill. While the worst cases can easily be explained "as too far gone to respond," many failures have been due to inept correction of the malocclusion although the periodontal care may have been excellent.

The face, jaws, and the musculature change when subjected to stresses. The periodontal membranes and bone break down when the stresses are not well distributed. The use of the flat plane can introduce poorly distributed stresses unless used with a well-trained and well-organized concept of tooth movement. The flat plane is of primary use as an adjunct during a transitional period of treatment when the *teeth are locked* into a malfunctioning position. The treatments of choice when the bite is locked into a malfunctioning position are reshaping of the teeth and/or correction of any existing malocclusion. The use of the flat plane to place all occlusal stress on the lower anterior teeth in order to "rest" the others is a peculiar concept since the body works best in function. The use of the flat plane to "free" the occlusion projects the human body and face as a static mechanism rather than as a biological mechanism. The use of a form of dental splint or retainer to distribute the occlusal stress is a much sounder concept.

The "passive eruption of teeth" on a flat plane is a subject for experimental rather than clinical research if the mechanism is to be recommended as a blanket form of treatment rather than as a device to be used in the mouth of a specific patient.

However, the correction of malocclusion, through use of the prosthetic appliances familiar to the hands and minds of the men using them, can be very simple and effective and should be encouraged. The following are examples of this use:

1. The use of partial dentures with labial wires, finger springs, bite planes or inclined planes.

2. The use of a fixed bridge and finger springs to jump a cuspid or lateral.

3. The use of two crowns or inlays to erect teeth and open a space by means of a loop spring, recurved spring or two nesting tubes and coil springs.

The advantages to the general practitioner or periodontist in the use of prosthetic appliances for the correction of malocclusion are:

A. Familiarity with the appliance.

B. Great stability of anchorage.

C. Tooth borne and/or supported.

D. Great leverage with very few harmful or deleterious forces. By and large, in well selected cases, the counterforce to the movement aids in correction of the malocclusion as in the use of two crowns with tube and coil springs for opening a bridge space.

E. Safety, since there are no semifixed or fixed connections.

F. Permanent retention, as in opening the bite with upper and lower partial denture.

A good functional occlusion can be established with the elimination of occlusal interferences. Relatively simple appliances, used in accordance with a comprehensive diagnosis, based on competent periodontal and orthodontic training and skill, can obtain excellent results in a great number of periodontal cases in which a simple malocclusion is one of the etiological or complicating factors. A primitive appliance used with primitive orthodontic skills can be successful only occasionally in the elimination of occlusal interference. An incomplete evaluation of the factors involved in the correction of the malocclusion in a specific mouth can lead to catastrophic results.

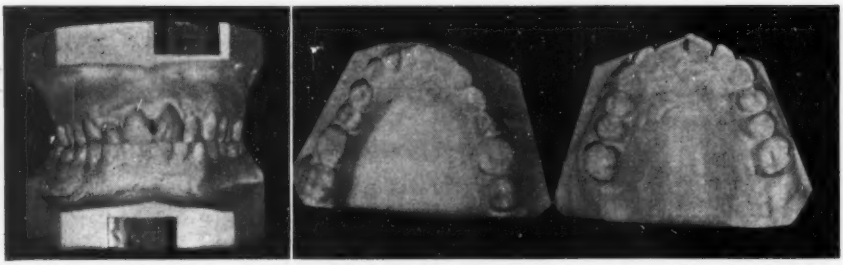

FIGURE 2 — Case I

Completed treatment—a Hawley appliance with two Crozat clasps was worn for two months. The upper centrals and laterals were brought into good functional articulation with mesio dental contact. The teeth were reshaped for better function and aesthetics. Food impaction and retention were eliminated and the teeth stabilized. Following active treatment, the patient was instructed to wear the Hawley appliance during sleeping hours.

The cases treated will usually conform to one of the following general categories:

1. Individual tooth movement or where there is a cross bite in existence.

2. The closed bite problem is perhaps the most perplexing problem of general restorative dentistry today, and also one of the most trying to treat. This is not because the tooth movement is so difficult but because retention is precarious. The treatment of individual tooth rotation and movement generally does not require intervention afterward by a periodontist or crown and bridge man, but closed bites invariably require persistence in treatment by both. Of course, there are certain cases where correction is not indicated in a closed bite.

3. The third type of case is one where it is impossible to insert prosthetic appliances, either removable or fixed, without movement of intervening teeth or dental structures. Simple tooth movement in these cases can make possible the insertion of a satisfactory chewing apparatus. In this type of case, there is generally deep periodontal involvement and the return to an ideal occlusion is neither indicated nor expected. It is to be desired, of course, but hardly ever possible, owing to the widespread destruction that has occurred in the preceding years. This type of case will generally show very poor mouth hygiene.

Subgingival curettage is indicated before orthodontic treatment is started. With the insertion of an appliance, the teeth can be brought into a good functional position. A fixed or removable prosthetic appliance will keep the teeth in position as a retainer. Without retention, a case of this type will collapse. Generally, in cases of this sort, particularly where the periodontal involvement is great, appliance therapy is very simple since very little movement is required.

ORTHODONTIC TECHNIQUE

Fundamentally, the most important factor in the correction of malocclusion is proper diagnosis. As treatment progresses, it may change. Appliances, too, may change. However, the establishment of the diagnosis is the basis of treatment. The therapist detemines what the face requires, where the teeth must be placed, where the jaws must be placed, and then determines how to do it.

Basically, the orthodontic appliance is a contrivance consisting of two major divisions, the fulcrum and the lever. The bands in the mouth and particularly the molar bands, are generally established as a fulcrum. The wires or arches are the levers. Bands are placed strategically on the molar teeth, and any other teeth, depending upon need and technique. In the labial technique, bands are placed on the molar teeth. A labial arch is inserted and the individual

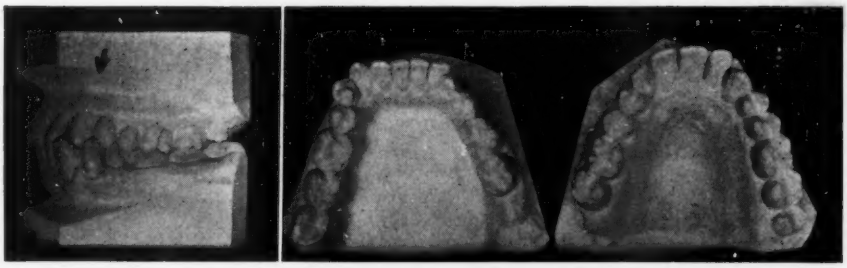

FIGURE 3 — Case II

Before treatment—Patient was a 46-year old male who had lost a permanent upper cuspid with no replacement and subsequent closure of the space. As a result, a crossbite relationship of lower left cuspid and first bicuspid with the upper teeth developed causing a locked bite with injurious lateral stresses. Food impaction, pocket formation and loss of alveolar bone resulted.

teeth which are required to be rotated or tipped are ligated to the labial arch. When a lingual is used, as in the Mershon technique, the labial or lingual arch is the lever and the teeth are individually ligated or moved by expansion or contraction of the lingual arch by the finger springs or by ligation to the labial or lingual arch, or by use of elastic bands in various arrangements. The multiband techniques are simply variations of the labial-lingual technique.

TREATMENT OF CASES

Cases I and II show the use of Hawley appliances in the correction of simple malocclusions. Case I, a thirty-eight year old female shows extreme protraction of the upper lateral incisors. The teeth were extremely mobile. Underfunction of the anterior teeth weakened the periodontium. Food impaction and retention were rampant. The etiology appears to have been a lip habit since the lower lip rested lingual to the lateral incisors, but labial to the central incisors.

A Hawley with a labial wire and two Crozat clasps was inserted. The spaces between the laterals and centrals were closed after two months of active treatment. The anterior teeth were reshaped and the patient advised to wear the appliance during

Completed Case—Treatment consisted of extraction of the lower left 1st bicuspid and construction of a bite plane carrying a labial wire wih two clasps. The labial loop brought all of the lower anterior teeth into position, closing the space left by the extraction of the bicuspid. Food impaction and traumatic occlusion were eliminated. The case is self-retaining.

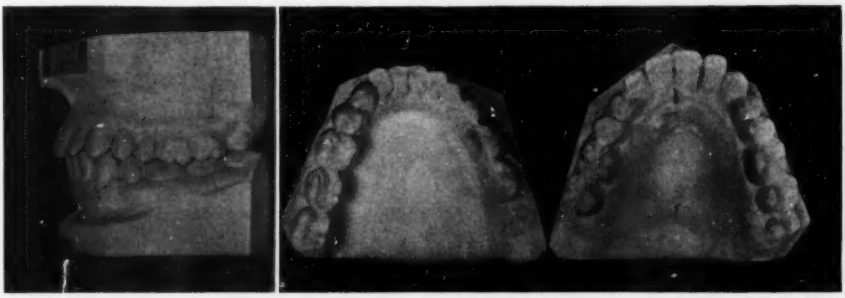

FIGURE 4 — Case II

sleeping hours as a retainer. The abnormal mobility of the anterior teeth ceased one month after treatment was completed. Healthy physiologic occlusion was restored, the teeth had mesio-distal support and all food impaction eliminated.

Case II shows a crossbite relationship of the lower left cuspid and first bicuspid with the upper teeth. The etiology was the loss of a permanent upper cuspid with no replacement and subsequent closure of the space. The mandible was forced into a protracted position. The patient was a 46-year old male.

The lower left first bicuspid was extracted. A bite plane carrying a labial wire with two clasps was inserted. The upper cuspid was shortened by reshaping but it rested on the bite plane. The labial loop brought all of the lower anterior teeth into position, closing the space left by the extraction of the bicuspid. The completion casts show the mouth after the completion of treatment, but before reshaping the teeth. The case is self-retaining. Again, a wholesome occlusion resulted with the teeth in mesio distal contact and all trauma eliminated.

CONCLUSION

Correction of malocclusion in the treatment of periodontal disease is accomplished under three general forms of treatment:

1. Correction of malocclusion through reshaping of the teeth by the periodontist.

2. Correction of malocclusion through the use of finger springs, inclined planes and other devices on prosthetic appliances by periodontist with orthodontic consultation.

3. Correction of malocclusion by the orthodontist through use of classical orthodontic appliances.

Occlusal interferences can be eliminated and a good functional occlusion established in a great many seemingly hopeless cases. Coupled with reshaping of the teeth and splinting further periodontal deterioration can be checked, but this can be done only in accordance with the principles which our great profession developed and our specialties evolved. Dummett summarizes the need for correct orthodontic procedures in the etiology of periodontal disease as follows:

"The correct form and position of the teeth will protect the gingivae from the traumatic irritation of mastication, will help to keep the cervical areas clean, and will aid in the maintenance of proper occlusion and sound alveolar support. In a significantly large number of cases, periodontal disease may be initiated by an irregular alignment of the teeth. The interdental tissues are squeezed by the crowding of the teeth, and food and oral debris are retained in the proximal spaces which are difficult to keep clean. The blood circulation of these tissues becomes impaired and there is likely to occur a gingival inflammation with or without hyperplasia.

"Other malocclusions such as excessive over-bite, mandibular protrusion and improper faciolingual position may produce similar inflammatory conditions."

All too true today is the statement by William Imrie in the *Parents Dental Guide* in 1834. "The means resorted to by different practitioners in regulating distortions of the teeth are various. A knowledge of the principles connected with the teeth is only to be gained by long experience, and the dentist, however ingenious he may be, who is not acquainted with these principles, will be constantly foiled in his endeavors to rectify deformities, while the patient meets, an increase of the malady. The principal methods in use are extraction of one or more of the irregular teeth; pressure by means of springs attached at their extremities to other teeth, assisted by ligatures of strong silk; a variety of gags; there is also the wedge, a simple and effective instrument."

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Periodontal Curettement in the Premaxilla

by JOHN I. INGLE, D.D.S., M.S.D.,* *Seattle, Washington*

PERIODONTITIS and traumatism involving the dental structures of the premaxilla pose a vexing problem to the periodontist. Having encountered a *severe,* generalized, horizontal destruction of the alveolar process in this region (Fig. 1) the clinician has often felt that his treatment, regardless of the method chosen, left much to be desired. He has customarily followed out a plan of treatment involving either (a) gingivectomy, with tissue removal to the depth of pocket, (b) odontexesis, which reduces the obvious inflammation, but which does not bring about any great reduction of the pocket depth, or (c) extraction of the involved teeth, with prosthetic replacement.

Of these alternatives, extraction is by far the most discouraging, nor does it always resolve the matter. Frequently, loss of the upper six anterior teeth leaves no choice but a full arch extraction with full denture prosthesis.

Fortunately a large percentage of cases need not be resolved by extraction. Where pocket depth is not great, odontexesis is often the indicated treatment, and when coupled with careful oral hygiene, will in itself guarantee success. In fact, odontexesis might well be initially employed under all circumstances so that localized inflammation may be reduced and tissue response evaluated. Of course, where pocket depth is too great, odontexesis alone will prove ineffective. Such cases are usually resolved by gingivectomy.

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In the premaxilla however, surgical gingivectomy carries with it two very serious drawbacks:

a. The gingivectomy patient who is constantly "before the public" suffers ten to fourteen days of mental discomfort while an unsightly surgical dressing remains in place. Moreover, even after removal of the dressing, the incised tissue still appears angry until the epithelial covering and normal gingival architecture are reestablished. (This problem may be solved in part with the plastic gingival veneer as suggested by MacKenzie.[1])

b. There is a definite traumatic shock to the psyche of many sensitive patients when they are faced with the realization that an obvious strip of tissue is to be excised from this esthetically important region. Some patients will balk at the mere thought of the operation, and will retain their disease rather than submit to the surgery.

SURGICAL CURETTEMENT

Where it is anticipated that neither extraction, odontexesis nor gingivectomy will yield the desired result, it has been found expedient to employ a certain method of *surgical curettement*. Properly performed, this procedure assures the following six advantages over the previously discussed methods:

a. Full visibility of all underlying structures is afforded through the employment of complete tissue retraction.

b. All the involved teeth are retained and a normal depth of sulcus is created where a pathological pocket previously existed, and would remain if odontexesis alone were employed.

c. There is no obviously marked and immediate change in the appearance of the gingival architecture following operation, because no gingival tissue is removed. Thus the patient is spared the psychic shock which accompanies radical gingivectomy.

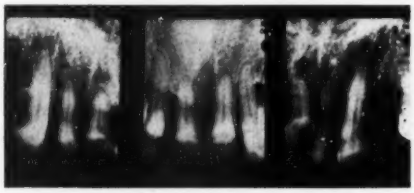


Fig. 1. Initial radiographic study. The severe, generalized, horizontal loss of the alveolar process in the premaxilla was produced by traumatism from occupational bruxism.

d. Following treatment, the gingiva recede to their new physiological level at such a slow rate that the patient unconsciously becomes familiar with the new gingival level and elongated crown.

e. Because no unsightly surgical dressings are used, it is not necessary to construct a plastic veneer as a means of camouflage.

f. Discomfort is greatly reduced.

CASE REPORT

The technique of surgical curettement was indicated in the case of Mr. J. S. G. who presented with severe periodontal destruction in the premaxillary region caused by traumatism (Figs. 1 and 2). Correction of the occlusal etiologic factors was initiated concurrently with the surgery.[2] Odontexesis was performed prior to the surgical treatment with a view to reducing the

Fig. 2. Clinical photograph taken at the first appointment. Note the extreme overbite, the localized recession and severe inflammation present. Reduction in the depth of pocket and removal of the traumatic forces are necessary.

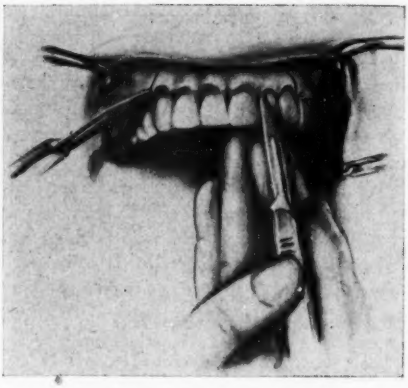


Fig. 3. The major incisions are made labial and lingual, just distal to each cuspid (left). A Bard-Parker #11 blade is used to incise across each interdental papilla (right).

inflammation. At a subsequent appointment the surgical operation was completed under infiltration anesthesia.

SURGICAL PROCEDURES

1. An incision approximately 10 mm. in length was made on the labial and lingual just distal to both upper cuspids (Fig. 3).

2. Each interproximal papilla was incised through its tip, thereby separating the labial and lingual tissues (Fig. 3).

3. A flap was retracted on both the labial and the lingual, exposing the roots of the teeth and the alveolar process (Fig. 4).

4. All of the chronic inflammatory tissue and the epithelial pocket lining was curetted from the region. The roots of the teeth were thoroughly cleaned and smoothed with both curettes and files. A thorough irrigation of the area followed (Fig. 5).

5. The "scalloped edge" of the marginal gingiva on both the labial and lingual flaps was reduced about ½ mm. so that the tissue would adapt more closely to the roots of the teeth (Fig. 6).

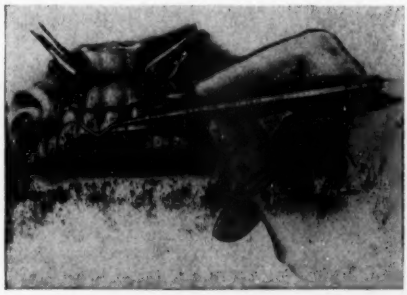
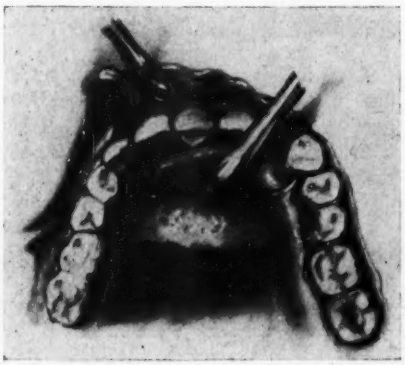


Fig. 5. Complete curettage of the underlying structures. The granulation tissue and epithelial lining are thoroughly curetted and the roots of the teeth are curetted, filed and polished. A Molt retractor #2 is shown.

Fig. 4. Retraction of the labial and lingual flaps. An 88W #7 wax spatula serves as a periosteotome. Care must be exercised to retract the periosteum along with the soft tissue.



6. The tissue flaps were returned to position and sutured securely with a "serpentine suture" (Figs. 7 and 8). The blood clot which forms beneath this tissue is the basis for the final result in the case.

7. The area was covered by protective tinfoil overnight (Fig. 9). Penicillin troches were prescribed for 48 hours to minimize the possibility of infection of the organizing clots. Ice packs were also prescribed, to reduce the local swelling.

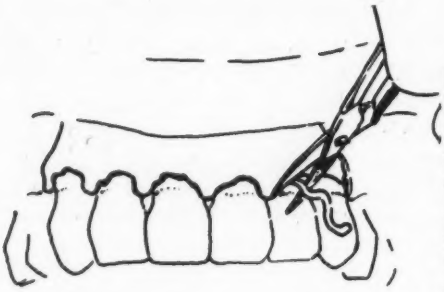


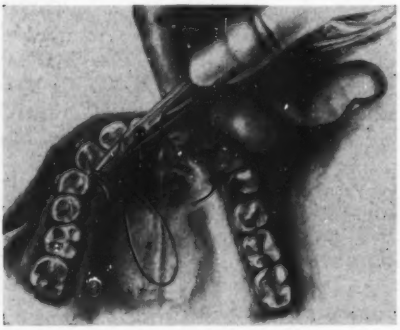
Fig. 6. Removal of the epithelialized margins from both the labial and lingual flaps. Full curved iris scissors or fine cuticle scissors are ideal instruments for this purpose.

8. After 48 hours the sutures were removed and the area gently cleansed of materia alba.

9. By the tenth postoperative day the tissues had retracted to their new level.

The patient (Mr. J. S. G.) has been seen on a six months recall regimen for the past two years. The present gingival sulcus ranges from one to two mm. as compared with a pocket depth of 4 to 7 mm. before surgery, and the tooth mobility has been reduced from third degree to first degree. The case has an excellent long range prognosis (Fig. 10).

Fig. 7. Clot is retained by suturing the labial and lingual flaps to position. An atraumatic ophthalmic needle mounted with "000" silk suture is used.



DISCUSSION

Reattachment of cementum to alveolar bone proper by new periodontal membrane fibers is possible if the three elements, cementum, alveolar bone and periodontal membrane are to be found in the *immediate* area of the desired reattachment. In a periodontal case where there is severe *horizontal* bone loss and deep pockets, and where the alveolar bone has resorbed to a depth of four to eight mms. below the crest of the gingiva, reattachment is out of the question. The only choice then open to the clinician is to artificially or physiologically

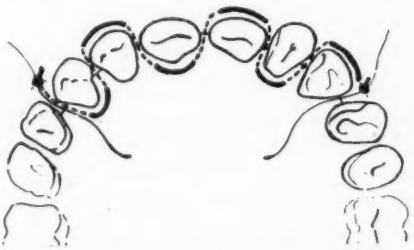


Fig. 8. "Exploded" view showing the pathway of the continuous "serpentine" suture. Note that the suture begins at the labial incision distal to one cuspid and then cuts through each interproximal space. (Demonstrated by the dotted lines.) The suture lies in the free gingival groove alternately on the labial and lingual. This groove affords retention for the suture, which, in turn, contours the gingiva by a "purse-string" action.

induce recession of the gingiva to a level just above the crest of the alveolar process, thereby allowing the patient to clean an otherwise unapproachable area.

A pocket depth of approximately three mm. can often be reduced by merely removing the inflammation from the area with displacement packs, odontexesis and thorough oral hygiene. However, if the depth of pocket has passed the point where the tissue will recede normally, the clinician must resort to some type of surgery. Too often gingivectomy has been performed in these cases, whereas more often than not, *surgical curettement* is the indicated procedure.

The tissue which is removed during an operation of the latter type is typical chronic inflammatory tissue characterized by round cell infiltration (Fig. 11). Ramfjord has shown that as long as such chronic inflammatory tissue exists in the area there will be no new connective tissue formation.[3] However, if the chronic inflammatory tissue is removed, as in the curettement, and its place taken by a blood clot, new connective tissue organization will rapidly occur. The speed with which a new cell-fiber pattern develops within the clot is well demonstrated in Fig. 12. Surprising as it may seem at first glance, the beautiful connective tissue fiber growth revealed in Fig. 12 *has developed*, and can repeatedly be shown to develop, *from a beginning blood clot in only eight days time.*

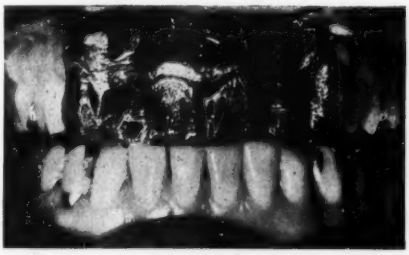


Fig. 9. Tinfoil protection here illustrated on a similar case. This protective coat may be prepared by painting tinfoil with compound tincture of benzoin and, while the foil surface is still wet, sprinkling it with denture powder.

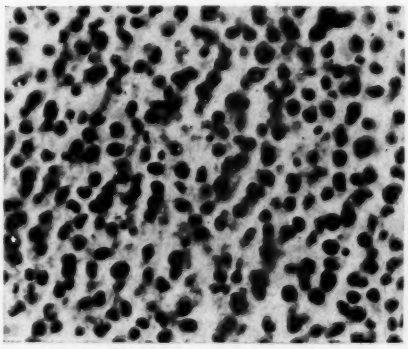
The normalcy and strength of the connective tissue which will evolve from a clot following surgical curettement is the key to the success of this type of operation. The fibrous connective tissue developing in an area of operation accounts for the new *firm character* assumed by the gingiva. Furthermore, the *physiological recession*, which slowly becomes apparent, is simply a manifestation of the fibrous retraction which characterizes any repair when normal connective tissue replaces inflammatory tissue.

Fig. 10. Clinical photograph taken two years after treatment was completed. Note the physiological recession and gingival architecture which has developed. The small inflamed area on the labial gingiva of the right central incisor responded immediately to odontexesis.

Before such a procedure as surgical curettement is undertaken, the patient should be made aware of the fact that *recession* of the gingiva is *certain* to occur, and is actually the only alternative to extraction. Those patients who are interested in retaining their teeth will readily consent to the surgery. When gingival recession and its accompanying crown elongation presents an esthetic problem, the condition may be alleviated somewhat by a judicious shortening of the upper anterior teeth or by the use of a plastic veneer gingiva.[4]

Two aspects of the curettement procedure deserve reemphasis. First, the epi-

Fig. 11. Granulation tissue taken from an area of chronic inflammation such as herein described. The preponderance of round cells is characteristic.



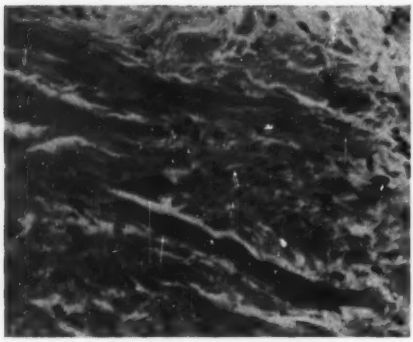


Fig. 12. Rebiopsy specimen taken 8 days later from the same area as shown in Figure 11. The new pattern of fibrous connective tissue has developed from a blood clot in this short time. It is the retraction of this tissue which produces the desired recession of the gingiva.

thelium which lines the underside of the labial and lingual tissue flaps and the chronic inflammatory tissue between the teeth must be entirely curetted away. Second, the very edge of the marginal gingiva[5] on both the labial and lingual must be removed with curved scissors.

By careful execution of the flap curettement and gingival trim, the epithelial lining of the old periodontal pocket will be so thoroughly destroyed that it cannot interfere with new connective tissue formation.

CONCLUSIONS

1. Gingivectomy as a periodontal treatment for severe, horizontal alveolar destruction in the premaxilla is esthetically and psychically traumatic to many patients.

2. *Surgical curettement* in the upper anterior region has many advantages over gingivectomy. The simplicity of the operation, coupled with the esthetic post-operative appearance of the area, and the absence of the psychic trauma engendered by other methods, would highly recommend this type of surgical operation to every periodontist.

3. The entire procedure of surgical curettement is described and illustrated.

4. The physiological recession which follows curettement is caused by retraction of the connective tissue organizing from the remaining blood clot.

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A theoretical discussion of some of the fundamental factors concerned with the

Development of the Periodontal Lesion

by JAMES NUCKOLLS,* *San Francisco, California*

WITH the development of microscopic technics which could be applied in the study of dental tissues, the fine morphological details of the normal structures of the periodontium have become evident. From these studies the foundation for the scientific study of periodontal pathology has been laid. The morphological descriptions of periodontal lesions are of practical aid to the periodontist, but classifying these lesions into various categories is the necessary first step in their understanding. It is also recognized that the terminology of the morphological pathologist should be the basic language of the field of oral pathology, for this has always been the privilege of taxonomy. However, I am not an advocate of complicated and unwieldy classifications of periodontal pathology; rather, I am more interested in tissue reactions to injury and the fundamental nature of the disease processes which affect the periodontium. Thus in my opinion the subject matter of the periodontium, like that of general biology, can be roughly divided into two parts: the static or the morphological, and the dynamic or physiological. Likewise, periodontal disease can be approached from the static or histopathological and from the dynamic aspect of an altered metabolism which results in a biochemical lesion. Knowledge of the latter demands as an essential prerequisite a knowledge of the former. It is therefore an encouraging situation when one finds in the literature that some of the researchers in the field of periodontal disease are driving further and further into the processes which precede morphological alterations in tissues.[1] Notable among these are the investigations of Glickman[2] on the measuring of oxygen uptake in healing periodontal tissues by the means of the Warburg technic, and those of Berg and co-workers[3] on glycogen determinations in inflamed and normal periodontal tissues.

Periodontal disease can be better understood through a working knowledge of carbohydrate metabolism, protein chemistry, and bone mineralization. A knowledge of these sciences and their interrelationship in health and disease can be applied to the periodontal tissue, clinical periodontal pathology, and to the biochemistry of periodontal lesions.[4] If the preceding can be accepted as a basis for discussion, it may then be assumed that basically similar tissues have similar physiological reactions in normal metabolism and react to alterations in their metabolism in a similar fashion. The periodontal tissues are no exception to these common biochemical mechanisms either in health or disease since they must react as do other like tissues in the human system.

*From the Section of Oral Pathology, Division of Preclinical Sciences, University of California, College of Dentistry. Presented before the American Academy of Periodontology, Oct. 11, 1951.

*Deceased, April 30, 1952.

Thus in our rationale we are able to extend our reasoning a bit further to the primary unit of tissue or the single cell. We will have gone a long way in our concept of periodontal disease if, as practitioners of clinical periodontology, we are able to extend our knowledge from the gross clinical lesion to the microscopic, and then to a realization of the importance of the biochemical lesion.[5] Likewise, the researcher in periodontal disease must regard the biochemical lesion as being the basis for all morphological alteration of tissue. It is my opinion that the time is past when the tissues of the periodontium are to be set apart from other similar tissue in the human system. This concept becomes especially apparent when an attempt is made to understand something about the basic patterns of living tissues and the fundamental metabolism of the functioning cell.

BIOCHEMICAL LESION

If one is to assume that the biochemical lesion precedes the microscopic and the physical lesion in disease, one must also be aware that a branch of science so young and incomplete as the biochemistry of disease must necessarily be rife with speculations and scanty of facts, and contradictory in nature. Although I have taken much of this material from the scientific literature not usually associated with periodontal disease, I have tried to avoid the more controversial topics. When this has not been possible, I have tried to give a critical account of the facts, but at the same time to speculate a little. I cannot wholly subscribe to the doctrine that speculation is out of place in scientific literature, for there are many gaps in this subject and unless these can be bridged, it is difficult or impossible to give a coherent account. My experience as a teacher has been that coherence is essential in elementary expositions. Speculation plays, and has always played, an important part in the advancement of scientific knowledge, for no research worker grasps blindly after something he knows not what; but he invariably begins with certain reasonable possibilities in mind. In short, he speculates. However, to speculate unreasonably and without achieved guideposts is worse than not to speculate at all. Another danger is that speculation is not always accepted as to its purpose, and therefore I will try to distinguish clearly between hypothesis and fact, hoping in this way to steer a middle course between unbridled imagination on the one hand and the equally undesirable fixation of traditional hypotheses and concepts on the other.

Probably one of the most controversial subjects in periodontology has been, and is, etiology.[6] Both local and systemic influences have been considered to be the sole factor. Both concepts have had their enthusiastic adherents. The consensus now seems to be that periodontal diseases may result from multiple complex factors. These may be general, or metabolic, local or irritational and infectious. Certain general predisposing factors may tend to lower tissue resistance. A tissue thus weakened may be subjected to a local precipitating factor and thus the probable causation of the local manifestation of a disease.

ETIOLOGY

In order that undue confusion may be avoided, I have purposely separated the local from the systemic causes in the development of the pathological changes in periodontal tissues.[5,7] In discussing these changes I shall attempt to discuss mechanisms from the standpoint of what seems to me the more important—or in terms of the basic processes that are the cause of the physical alteration. Because of the brevity of the paper, disturbance of systemic mechanisms which may influence periodontal disease will be referred to only as examples. This is done with the idea of pointing out that both local and systemic dysfunctions produce pathological changes in the periodontium, each operating in a different way to produce characteristic changes in bone,

connective tissue and epithelium.

Before taking up the more detailed microscopic and biochemical phases of tissue changes resulting from an altered metabolism, I should like to briefly discuss a concept of the gross development of the local periodontal lesion and the formation of the pathological pocket.[4, 5, 7] The onset of the lesion is initiated by an irritant which elicits an inflammatory response on the part of the investing tissues of the periodontium.[5, 8, 9] This reaction may range from a barely perceptible reaction or a hyperemia to the production of an abundance of pus and a loss of tissue. The irritant may be trauma of various kinds such as mechanical irritation, or calculus, or the byproducts of bacterial metabolism, or any agent which would produce tissue injury. The overall histological picture of the periodontal lesion is characterized by a progressive inflammatory involvement of the connective tissue, epithelium, and bone of the alveolar process. If the stimulation is violent, as in the case of acute ulcerative gingivitis (Vincent's infection), a quick inflammatory reaction is provoked. There may be a rapid breakdown of the peripheral periodontal tissues. The toxic byproducts of bacterial metabolism and of tissue breakdown[10, 11] may produce a marked inflammatory reaction and diffuse resorption of the crest of the alveolar bone. When the condition is slowly progressive and extends over considerable periods of time, the gingival tissues present a clinical and morphological picture of chronic inflammation. There may be an accompanying marginal osteitis at the height of the alveolar crest, although the teeth appear to be firm in their sockets. The architecture and the trabeculi of the supporting bone beneath the rarefied area often appear normal in the roentgenograph. The end result then is a prolonged pathological process in the development of which complex alterations take place in the local structure, oftentimes resulting in pocket formation and the loss of teeth. In a sense, an open lesion of inflammatory origin is established which—depending on the character and persistence of the irritant—may be either acute, subacute, or chronic in nature. This lesion is then a raw lesion which, in the more acute phases, is partially or completely denuded of epithelium. Hemorrhage is almost spontaneous. The collagenous fibers of the connective tissue are swollen and edematous, and the tissue elements are masked with inflammatory cells.[4, 12]

EXTENSION OF LESION

In the acute phase of inflammation the reaction extends along the gingival perforating vessels from the connective tissue convolutions between the rete pegs of the epithelium to the crest of the alveolar process.[13] Depending on the character of the lesion, the alveolar crest undergoes either horizontal resorption with equal loss of the alveolar crest, or a vertical resorption which may include only one side of the cortex of the tooth socket. As the pocket develops there is first seen a proliferation of the basal cells of the epithelial attachment out into the connective tissue. At this point the epithelial cells may tend to proliferate and extend down the cementum of the root surface. However, this initial proliferation along the root surface occurs more often in the first phases of gingival inflammation. If the irritant is persistent and severe, the end result is the solution of these tissues and an open lesion is formed.

Once an open lesion is established and the underlying connective tissue is denuded of its epithelium, the pathological pocket heals by forming granulation tissue. The granulation tissue partially replaces the lost connective tissue by proliferation. The affected part undergoes an organization which results in a functional repair but never a complete regeneration to the original form. If there has been a resorption of the alveolar crest, there may follow a concomitant reduction of the associated structures and the root of the tooth is exposed.

I should like to emphasize, at this point,

that it has been my observation that in the early phases of gingival inflammation there is a tendency for the basal cells of the epithelial attachment at the cemento-enamel junction to proliferate down the root. However, if there is a loss of tissue, the repair of the wound follows the normal course of wound healing. This includes the removal of the irritant and the development of a connective tissue stroma on the soft tissue side of the pocket. This stroma first appears as granulation tissue, and should not be confused with overproduction which is associated with chronic irritation in tissue healing. The latter should be more correctly referred to as granulomatous tissue. Although similar in nature, the granulomatous tissue of inflammation differs in certain respects from the granulomatous tissue of reticulo-endothelial diseases.[14]

As tissue organization of the wound continues to develop a connective-tissue stroma, epithelial cells proliferate inward from the periphery of the wound.[15] The advancing epithelium eventually covers the surface of the healing lesion and gradually organizes into stratified squamous epithelium lining the surface of the pocket, forming an attachment with the cementum of the root. The periodontal pocket is now established with a complete epithelial lining. Thus the epithelium of the gingival sulcus does not creep down the root surface when there is an acute or subacute open periodontal lesion, but forms a stratified squamous layer covering the connective tissue which is undergoing repair in the process of wound healing.[4,5] For the purpose of contrast, in the preceding discussion, I have discussed two extremes in the devolopment of the periodontal pocket. This is done with the full realization that pocket formation is not a stereotyped process and that any degree of periodontal pathology intermediary between the two may occur.

A very slow progressive gingival recession, less dynamic in action, which accompanies certain atrophic conditions and age changes, may give the appearance of epithelium creeping down the root surface. These processes are of greater subtlety than the inflammatory reaction and may not be observed clinically except for their final result. These, however, may be systemic in nature and such tissue changes come about by a different mechanism than those of inflammation.

CALCULUS

Of all the local factors which have been incriminated as causes of periodontal inflammation, calculus probably plays the most important role. Calculus is generally recognized to be calcified organic material on the clinical crowns of teeth which is more abundant near the orifices of the salivary ducts. Calculus is also deposited along root surfaces of the pathological pocket. In view of the differences in locations of the calcareous deposits, as well as possible differences of mechanisms which cause its deposition, it would seem that an arbitrary differentiation between the two types should be made. I shall, therefore, discuss calculus deposition from the standpoint of oral calculus, or that which accumulates about the exposed crowns of teeth, and subgingival calculus, or that which is deposited deep within the pathological pocket. There seems to be enough general biochemical evidence at the present time to warrant a distinction between the two. There are certain differences in the processes that cause oral and subgingival calculus formation.

From the standpoint of the periodontist, both types of calculus—oral and subgingival—are a definite source of gingival irritation; but whether both gingivitis and calculus are the results of other factors is a matter of considerable conjecture because of a dearth of good biochemical data in this field. Even if abundant data were available and the exact processes which cause calculus formation were known, it is highly questionable that the formation of oral calculus could be controlled, since it is so intimately connected with general mineral metabolism.

There seems to be a consensus that a mechanism is operating in the oral cavity which produces a state of supersaturation with regard to calcium and phosphate before a precipitation of these salts can occur. However, the process by which a state of oversaturation is achieved is still a matter of discussion.

There are many hypotheses as to calculus formation which for the greater part are supported with evidence totally unrelated to known physiological mechanisms. In a recent publication on calculus formation Hodge and Leung[16] suggest that the proponents of the various hypotheses may be divided into three groups. There are those that hold that the microorganisms of the mouth—by their growth and metabolism —produce conditions which lead to the precipitation of the elements which enter into the formation of oral calculus. A more recent example of this kind of reasoning might be deduced from the work of Kesel[17, 18] and co-workers who propose that the mouths of certain individuals contain bacteria known as lactis aerogenes which break down amino acids of the saliva and produce ammonia and nitrogen. The presence of ammonia could then bring about an alkalinity which would favor the precipitation of calcium and phosphate from the saliva. This line of rationalization would account for calculus deposition and no caries, or, conversely, caries and no calculus, if dental caries is an acid decalcification.

Recently Ballantine, Clegg, Rae and Lawford,[19] studying ammonia formation in saliva, showed that the addition of 3 per cent urea caused an increase in ammonia production, indicating the presence of urease in the saliva. The presence of urease in the saliva might also be a factor in causing an alkalinity in the oral cavity, providing urea is an oral constituent.

Bacteria of the genera Leptotrichia and Actinomyces have been associated with calculus formation in that they are considered to produce a local alkalinity and a decomposition of salivary proteins in their immediate vicinity. The change in local environment, in turn, brings about a precipitation of calcium salts from the saliva.[20] The ability of these organisms to produce such changes, however, has not been clearly demonstrated. In vitro studies on actinomycetes recovered from tooth surfaces indicate that these organisms are capable of producing acids in some quantity when supplied with sugar media.[21]

ENZYMES

A second group of investigators favor the hypothesis that calculus results from an oral enzymatic mechanism. A concept of this kind is advanced by Rapp[22, 23] who postulates that there is present in the saliva an enzyme carbonic anhydrase. This enzyme catalyzes the breakdown of the bicarbonate ions which are constituents of the saliva. This breakdown liberates carbon dioxide and the hydroxyl ion. Because of the differences in partial pressures between freshly secreted saliva and expired air, there is a rapid escape of the carbon dioxide gas from the saliva. An increase in the hydroxyl ion concentration would therefore tend to alkalinize the saliva beyond the state of saturation leading to calculus deposition. The patients whose salivas contain the enzyme carbonic anhydrase have calculus and are considered by Rapp to be calculus positive, whereas patients whose salivas contain an anti enzyme (potassium thiocynate) which inhibits the reaction are calculus negative. This hypothesis receives some support from Baldwin[24] who points out that this enzyme has a wide distribution in animal tissues and catalyzes the splitting of carbonic acid to yield carbon dioxide and water, and in the reverse direction, the hydration of carbon dioxide, to yield carbonic acid.

The presence of the phosphatase enzyme in gingival tissues has led to another postulation. This concept presupposes this enzyme could react with certain organic phosphate complexes in the saliva to liber-

ate free inorganic phosphate which, together with calcium and other elements already present in the saliva, would precipitate out as calculus.[25, 26, 27] This is but one of several current hypotheses which suggest that phosphatases of the oral cavity may play a role in the deposition of salivary calculus.[28, 29] These concepts probably have their foundation in the early work of Robison and co-workers[30, 31] who were among the first to suggest that phosphoric esters and an enzyme played a role in the calcification of bone and cartilage. The enzyme is alkaline phosphatase and is responsible for the cleavage of inorganic phosphate from intracellular organic phosphate in the mineralization of bone, dentine, enamel and cementum.

Whereas the theories of physiological calcification have been greatly advanced in recent years, there seems to be little relationship between the biochemical mechanism of normal physiological calcification and any possible oral enzymatic process which leads to the deposition of calculus.

An analysis of the physical composition of calculus shows it to be quite similar to that of enamel, dentine, and bone.[16] The calcium phosphate crystals of bone, enamel, and dentine are described as having an hydroxylapatite lattice which refers to a definitive arrangement of calcium, phosphate, oxygen and hydrogen ions. Certain properties of the apatite lattice have also been reported for calculus. Since saliva is normally in a state of saturation with respect to calcium, phosphate, bicarbonate, and hydrogen ions, one would therefore expect to find enamel as well as calculus at a similar point of saturation. If the saliva was undersaturated, calculus would not form and there would exist the possibility of enamel decalcification. There is also another possibility that saliva may exist in a supersaturated state due to the presence of organically bound calcium and phosphate. Any factor that would tend to bring about a release of inorganic calcium or phosphate from its organic combination would tend to increase the saturation and favor precipitation. Another possibility is that the saturation of saliva may be increased by decreasing the hydrogen ion concentration through the loss of CO_2 which removes the acid element from the saliva.[32]

CALCULUS FORMATION COMPLEX

In any event when one speculates on the chief etiological factor in periodontal inflammation—the formation of calculus—the hypotheses which consider such factors as carbon dioxide loss, ammonia production, or the decomposition of organically bound calcium or phosphorus lead to one point in common: a decrease of the solubility of calcium and phosphate which results in the deposition of calculus. Normal saliva is either saturated or supersaturated with regard to its calcium, phosphate bicarbonate, hydrogen ion complex. The bicarbonate ion seems to play a major part in the formation of carbon dioxide gas. The loss of CO_2 from the saliva tends to produce an oversaturation of the saliva and the deposition of calculus. Whether the production of CO_2 is the result of a breakdown of the bicarbonate of the saliva by an enzymatic reaction as suggested by Rapp, or the decomposition of the bicarbonate by the addition of hydrogen ions to the bicarbonate-carbonic acid system are matters which must await further investigation. The possibility that other mechanisms such as ammonia production and decomposition of proteinates, as well as other factors which may enter into the precipitation of calculus, must also be given consideration. It is becoming evident that the formation of oral calculus may be as complex a process as that of dental caries.

The differentiation between oral and subgingival calculus is probably justified not so much because of the difference in mechanisms but because of the differences in substrates from which the calculus is derived. It is difficult for me to envision saliva infiltrating into a subacutely inflamed periodontal pocket of six or eight

millimeters in depth. This seems even less probable when one considers that there is a continuous flow of exudate and pus from these pathological pockets, yet calculus forms along the sides of the roots in these areas. If these pockets are subacute or chronically inflamed, the exudate which escapes from the permeable tissue and vessels contains all of the elements of the serum which forms the fluid basis of circulating blood. This serous exudate, which forms a part of the pocket's content, is a saturated solution with regard to calcium, phosphate and other ions. The presence of organically bound calcium and phosphate in the form of proteinates in the escaped serum, would further increase the possibility of an oversaturation to the point of calculus deposition. The CO_2 present in the body fluids also increases their ability to hold calcium and phosphate in solution.[33]

Any condition in the periodontal pocket changing the organically bound calcium and phosphate to the inorganic form would thereby increase the saturation of the exudate. Likewise, the escape of carbon dioxide from the exudate would also tend to produce a supersaturation to the point of calculus precipitation. The two types of calculus—oral and subgingival — are not essentially different in composition[16] and probably their essential mechanisms of deposition are similar. The loss of carbon dioxide in either instance may account for the degree of oversaturation which is essential to the formation of calculus.

When calculus formation is viewed from the foregoing discussion, it becomes evident that the mechanism of calculus formation differs markedly from the process of normal mineralization of bone, enamel, and cementum, which are essentially cellular processes. Viewed in this light, calculus then might be considered an ectopic calcification and similar to that of renal calculi, pathologic calcification of connective tissues, and like calcifications described in the literature. From the foregoing discussion, it would seem that the formation of calculus, both oral and subgingival, should be regarded as a pathological process, and as such marks the beginning of a series of local inflammatory changes which may result in the loss of teeth.

The inflammatory reaction[8, 9, 4, 5, 34] nowise differs in the periodontal tissues from that of other connective tissue and bone. The inflammatory response to an irritant, whether it be calculus or from other causes, is first evidenced by a change in capillary filtration. The cell walls as well as the local vessels become more permeable. As a result of the increased permeability of cells and capillaries, a serous exudate engorges the extracellular compartments. This change is followed by the precipitation of fibrin in the lymph channels, the capillaries and the extracellular spaces. As the result of fibrin precipitation, the normal oxygen supply by the way of the arterioles is cut off, and, in turn, the normal oxidation reduction reactions within the cells are prevented from going towards completion. The region develops a hydrogen ion concentration and a metobolism peculiar to the localized area. As a result of the irritant and the concomitant inflammatory reaction, the metabolic rate increases in the localized area resulting in a rapid depletion of available oxygen, and a state of local anoxia develops.

CO_2 CONTENT FALLS

The oxygen want in this area upsets the carbohydrate metabolism and as a result lactic acid[35] is formed instead of the normal end products of carbon dioxide and water. There is also a depletion of the cell glycogen from which energy is derived.[36, 3] The energy requirement of the functioning cell is then derived from protein of the exudate and a state of local gluconeogenesis ensues.[37] The CO_2 content of the inflamed area tends to fall because of the impairment of Kreb's cycle, and this is accompanied by a concomitant drop in the tissue buffer in the area. As cellular function continues, there is an increasingly greater

accumulation of lactic acid which is not removed by normal channels, and an uncompensated local acidosis develops. With an increasingly lower pH there is an accompanying change in the inflammatory cellular picture. At first there is an increase in the polymorphonuclear leukocytes. With a decrease in the pH these cells become fewer and the mononuclear leukocytes make their appearance. These appear to be fairly normal at a pH of 6.9 to 6.8. If the inflammatory process is unmitigated and the pH falls below 6.5, autolytic enzymes already present in the tissues may be activated by the acidity and a depolymerization of the tissue ground substance results.[38, 39, 40, 41] These early tissue changes may precede any alteration in the cellular picture and can only be determined by histo-chemical procedures.

As the inflammatory process extends along the gingival perforating arteries to the alveolar bone, the first roentgenographic evidence of bone involvement is seen as a break in the continuity of the cortical plate at the tip of the alveolar crest.[4, 5, 7] This may be interpreted as the establishment of a biochemical lesion and a progressive involvement of the deeper structures of the periodontium. If the irritant is persistent, as in the case of calculus, the marginal alveolar crest begins to show, roentgenographically, a rarefaction which, if allowed to continue, will result in a progressive loss of bone, pocket formation, and the eventual loss of teeth.

BIOCHEMICAL CHANGES

The biochemical changes in bone injury are essentially the same as those of inflammation in soft tissue,[42, 43, 44, 45] the difference between bone and other mesoblastic tissues being in the former's potential to aggregate mineral salts in a localized area.[42] This process will be referred to as physiological mineralization.

The reaction whereby bone salts make their appearance in the intracellular spaces of a bone matrix occurs by the catalytic action of an enzyme on a glucose phosphate substrate within the living cell.[46, 47, 48, 49] This enzyme known as alkaline phosphatase[30, 31, 50, 51, 47, 52] is found in the osteoblast, ameloblast,[47, 53, 54, 55, 56, 57, 58, 59] and odontoblast[60, 61] prior to the onset of calcification, and is present in the cell during calcification. The enzyme alkaline phosphatase is considered to act in a specific way bringing about a cleavage of glucose-6-phosphate in the cell[58] to liberate inorganic phosphate ions into the extracellular bone matrix. Thus both glycogen and phosphate[62, 63, 64] are concerned with the calcification process of bone and other hard tissues. Teleologically, the carbohydrate phase serves as an accumulator of phosphate. The cleavage of organic phosphate by the enzyme liberates inorganic phosphate ions in sufficient amounts in specific areas, which, with the calcium and phosphate ions already present in the extracellular fluids of the bone matrix produces a supersaturation of the salt. When a local supersaturation is achieved, there is deposition of the mineral complex in the bone matrix.

The bone salts[65, 66, 67, 68] are stable within the matrix only at a normal tissue pH of 7.4.[42, 69, 70] When inflammation tends to disturb the intermediary or carbohydrate metabolism in the cell, the result is the accumulation of lactic acid. With an increasing acidity in the localized area the bone salts become increasingly more soluble. These salts tend to go into solution leaving the matrix in varying degrees of denudation.

The roentgenographic picture of such a state shows a preipheral area of rarefaction gradually diffusing toward an area of normal appearing bone. Microscopically the picture is one in which the bone cells appear unmasked and surrounded by a decalcifying and disintegrating organic matrix.[71] With the persistence of the irritant, the inflammatory process is progressive, involving more and more of the periodontium until a pathological pocket is formed.

McLean and Bloom[72] speculate that both salt and matrix disappear together. When one considers the way in which biological changes occur, it is quite possible that both the bone salt and matrix are affected as the result of a low pH in the inflammatory area. These authors also mention that they have observed no evidence of phagocytosis of the bone salt by osteoclasts.

The theories of bone building and resorption are somewhat at variance with the older and traditional theories of osteoblastic osteoclastic action. The older hypothesis had its origin in the histological observations of Sir John Goodsir (1814-1867)[73] who first used the microscope in bone research and was first to observe the bone cell. To Goodsir a bone was a hive of bone corpuscles wrapped within a nest of periosteum. Each bone cell, therefore, was endowed with the power to build up or tear down that part directly under its control.

The biochemical and histochemical concepts of bone building and resorption have their beginning in the researches of Robison and Soames (1924-1930).[30,31] Since that time the foundation laid by these researchers has advanced from strength to strength as evidenced by the heavy documentation of this section of the paper.

Living systems carry on their activities by value of myriads of chemical reactions which are collectively referred to as intermediary metabolism. Apparently there is a different enzyme for practically every reaction. This can only mean that some several thousand enzymes exist in a single cell. The study of intermediary metabolism represents one of the oldest lines of biochemical investigation, and here the entire process from glucose to glycogen and from glycogen to lactic acid which has been shown to occur in the inflammatory process has been reconstructed in vitro and involves some twenty or more enzymes, and many of these enzymes have been prepared in pure state.[74]

The attempt to treat basic causes and to assist the physiological mechanisms of healing and repair will mark a new era of dentistry. The older methods developed without any knowledge of underlying reasons in their favor, often worked well; however, in the absence of any critical understanding on the part of the practitioner, it is frequently mere chance which leads to a favorable result. As these basic reactions become better understood and are applied to the various branches of dentistry, the vast possibilities of dentistry as a biological science now seen only dimly will emerge clearly and a new era of dental science will be at hand.

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(Continued on page 191)

A Radiographic and Histologic Study on Reattachment*

by FRANK E. BEUBE, *New York, N. Y.*

THERE is no unequivocal evidence at the present time that demonstrates reattachment. Many investigators, including the writer, have shown that new alveolar bone will form around teeth, where formally there had been considerable bone resorption with accompanying periodontal pocket formation. When this data was correlated to experimental studies in animals** it was concluded that reattachment had occurred.

Radiographic examination of cases, which revealed the deposition of new bone after periodontal therapy, usually disclosed a wide periodontal space between the new bone and the tooth (Figs. 1-10). It cannot be interpreted from the radiographs or by clinical probing, that these spaces contained new cementum and new periodontal membrane fibers or there lacked all connection between tooth and bone, and only an artificial space was present, indicating a narrow deep pocket. To determine whether the space between the treated teeth and bone showed new cementum and periodontal membrane it would be necessary to do histologic examination of the supporting tissues and roots of the treated cases. A clinical, radiographic and histologic report on one of these cases follows:

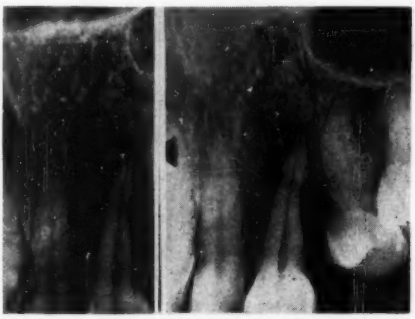


Fig. 1. Two views of marked resorption of bone around maxillary cuspid due to periodontal disease. Deep pockets were associated with the bone loss.

CASE REPORT ON G. K.

1. *Medical History*—prior to September 1933—Perinephritic abscess—3/19/32; essential hypertension; chronic arthritis of joints (infectional 7/25/32); Furunculosis 9/1/32; Lumbar hernia and left chronic nonpurulent frontal sinusitis 2/19/33.

ORAL HISTORY AND EXAMINATION

1. Loss of all posterior teeth without replacement.

2. Clinical examination of mandibular right canine showed a wide mouthed pocket extending from labial to lingual on mesial surface, 8 mm. in depth.

3. Radiographic findings disclosed a "V" shaped resorption of bone to the apical 3rd of the root. This was classified as an infrabony pocket (Fig. 11).

TREATMENT

1. Curettement of the root surface for the removal of the calcareous deposits, the epithelial and granulomatous tissue lining the pocket.

2. Occlusal grinding—limited in effectiveness because of overload to the periodontium of the twelve remaining teeth.

3. Home Care procedures—use of the toothbrush, rubber stimulator and bland mouthwash.

4. Flap operation with the insertion of

*Presented to the American Academy of Periodontology Oct. 27, 1950. From the Section of Periodontology, School of Dental & Oral Surgery, Faculty of Medicine, Columbia University.

**Frank E. Beube, D.D.S.—A Study on Reattachment of the Supporting Structures of the Teeth. Jl. of Perio. April 1947, P. 55 to 66.

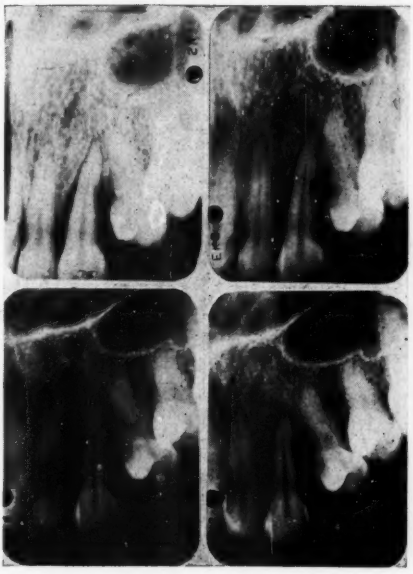


Fig. 2. Bone deposition has occurred around maxillary cuspid after periodontal treatment. (see fig. 1) But a wide periodontal space is present. The four radiographs were taken on the same day and exposed at one, two, four and six seconds respectively.

Fig. 3. A later postoperative period than seen in figure 2. The periodontal space is narrower. (Exposures were 1, 2, 4, and 6 seconds.)

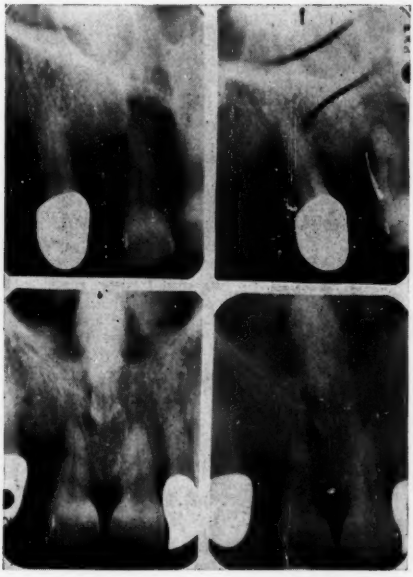


Fig. 4.

Figs. 4, 5, 6 & 7. These radiographs show the preoperative findings in cases with extensive bone loss and deep pocket formation from periodontal disease with accompanying radiographs showing the formation of new bone, but wide periodontal space remaining after treatment has been completed.

Fig. 5.

Same as Figure 4. The dates the X-rays were taken are as follows: 12-16-49; 1-17-50; 3-3-50; 6-27-50; 9-26-50; 9-26-50. The exposure time has varied in the last 2 X-rays.

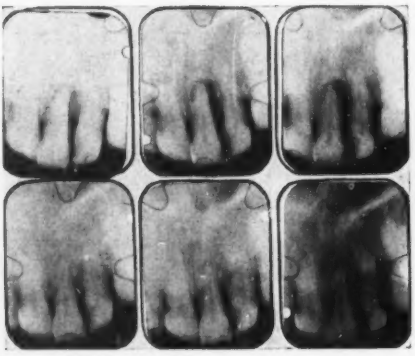


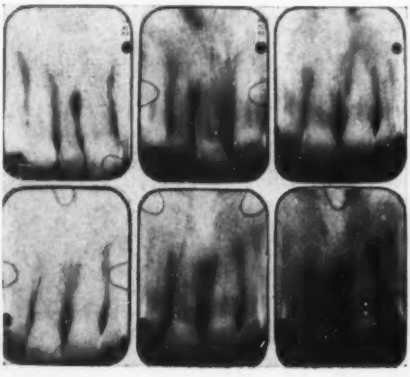
Fig. 6.

boiled cow-bone powder in April 1934 after failure to get pocket closure by curettage which was done intermittently from Sept. 1933.

5. Insertion of soldered castings for splinting of mandibular right lateral incisor and canine in 1936.

Fig. 7.

Same as Figure 4. The dates the X-rays were taken are as follows: 9-20-49; 11-30-49; 5-4-50; 10-18-50; 10-18-50; 10-18-50. The exposure time varied for the last 3 X-rays.



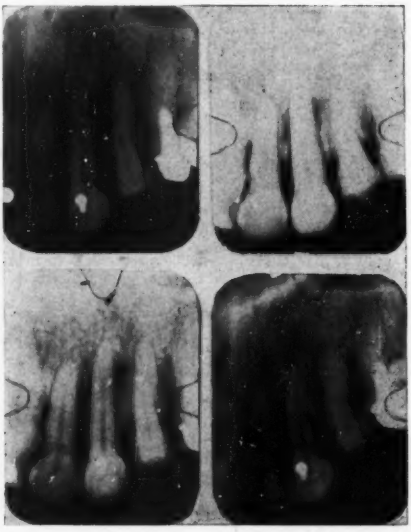
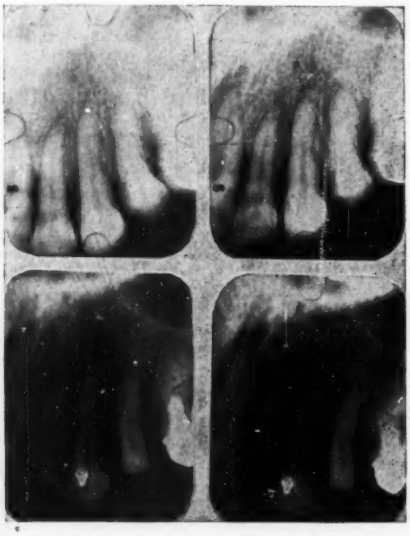


Fig. 8. Upper left radiograph taken 6/10/48, shows preoperative condition with extensive bone loss from periodontal disease. Deep pockets were associated with the bone resorption. The next two radiographs were taken 3 months postoperative and show deposition of new bone with wide periodontal space. The bottom right radiograph was taken one year later and the periodontal space is narrower.

Fig. 9. The four radiographs disclose narrowing of the periodontal space a few years postoperative of figure 8. X-ray exposures were made at 1, 2, 4 and 6 seconds respectively.



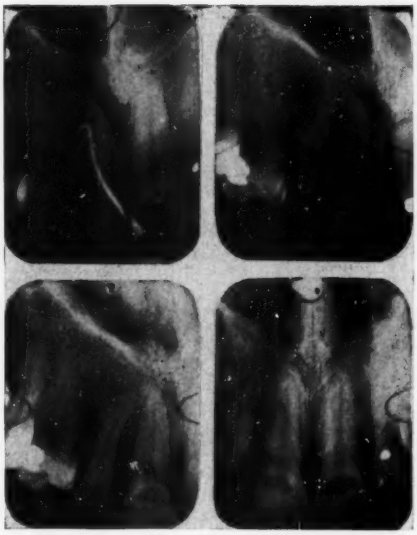


Fig. 10. The two upper radiographs show the preoperative appearance of periodontal disease around a central incisor. The gutta percha point roughly indicates the outline of pocket. The two bottom radiographs show new bone deposition and a narrow periodontal space 5 years postoperative.

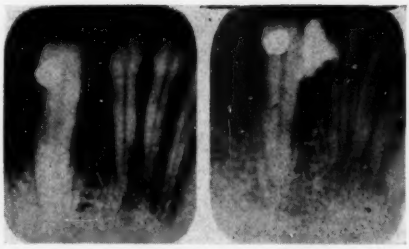


Fig. 11. The radiograph on the left was taken immediately preoperative and discloses extensive bone loss with deep pocket formation around the mandibular cuspid. The radiograph on the right shows the condition immediately postoperatve.

RESULTS

The operated area was probed and radiographed at regular intervals for two years. During that time alveolar bone was laid down steadily and no pocket could be detected (Fig. 12). A sulcus of about 2 mm. was always present. At the end of the two year period the lateral and cuspid were splinted together with two castings soldered at the contact areas. For the next 13 years, radiographs were taken at varying intervals. The new bone which had been laid down in 1936 was not only maintained but became denser through the years. A lamina dura had also formed (Fig. 12).

During the fifteen years of observation the patient refused dentures and had been losing one or two of his anterior teeth every few years, due in part to occlusal overload and resulting advanced periodontal involvement. The supporting structures about the

Fig. 12. One year postoperative Fig. 11. Some new bone has been deposited, which can be seen in radiograph on extreme left. The center radiograph and the one on the right show two different views 15 years postoperative. The crest of the bone appears denser and gives the appearance of a lamina dura.

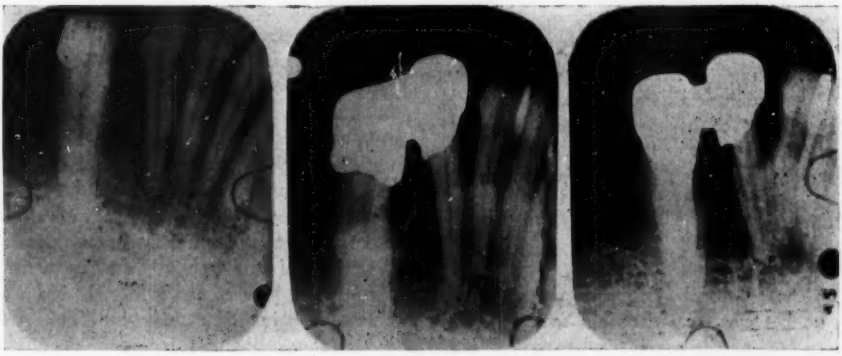


Fig. 13. Histologic section of case shown in figures 11 and 12. Note the debris immediately above the transseptal fibers, which are attached to new cementum on the root from N to C. Note dense bone immediately apical to transseptal fibres.

Fig. 14. High power of figure 13, where the new cementum was laid down and attached to transseptal fibers. Note irregular outline of old cementum and narrow space between new and old cementum.

mandibular right lateral incisor and cuspid remained intact. By July 1949 he had so few teeth remaining that he was advised to have full dentures.

On July 1949 the lateral incisor and cuspid were removed with a block of bone and new supporting tissues. This was placed in 10% formalin for fixation and histologic examination. The sections revealed that a new periodontal membrane and alveolar bone were present on the root surface where formally the radiographs and retraction of gingival flap disclosed the root to be denuded of bone. Transseptal periodontal membrane fibers had formed incisal to the crest of bone and attached to a new layer of cementum. Evidence of inflammation was noted in the gingiva and the gingival sulcus (Fig. 13).

DISCUSSION

As previously described the periodontal pocket was located on the mesial proximal surface of the cuspid, and extended widely from the labial to lingual tissues. This was confirmed prior to making the incision. The radiograph in 1933 disclosed two-thirds of the root length of the cuspid was denuded of alveolar process and periodontal fibers. That the loss of bone and periodontal membrane was extensive and to the apical third of the root and in a labial and lingual direction was verified by the open flap operation. The denuded area was filled with soft granulomatous tissue. The radiographs in 1948 demonstrated that the radiolucent area as seen in 1936 was filled in with new alveolar bone. It is significant to note that the histologic sections demonstrated that at whatever portion of the mesial surface that a section was taken from, the transseptal fibers of the periodontal membrane were attached to cementum at the base of the sulcus. It is likely that this point of attachment could not have existed so high incisally when the

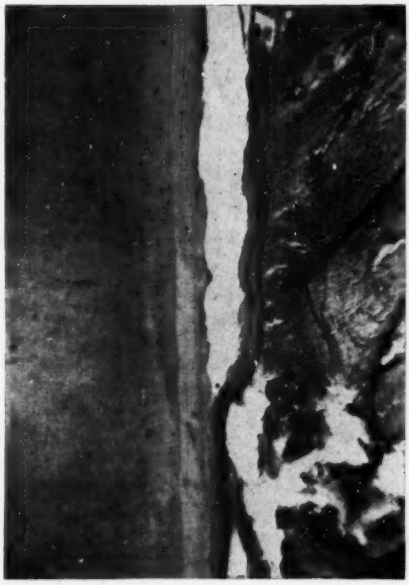


Fig. 15. High power view showing new cementum which had pulled away from old cementum, but remained attached to periodontal membrane fibers.

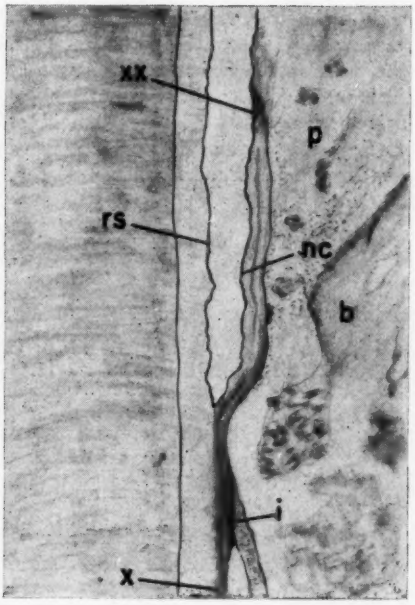


Fig. 16. Diagram of Figure 15. The most apical point reached in treatment is indicated by x. New cementum was formed up to xx. The root surface after scaling is indicated by rs. The injury to cementum posibly done by scaling at time of "pocket" treatment is indicated by i. New cementum nc. Bone, b. Periodontal membrane, p.

operation was performed. Because prior to any periodontal interference in 1933, the epithelial and transseptal fiber attachment must have been located at the apical third of the root. Another important finding is noted in the cementum. An earlier layer of cementum shows an irregular outline which could either be resorption or a roughened surface from the instrumentation during the operative procedure (Fig. 14). While removing the tissue block with mallet and chisel, the layer of cementum in contact with the irregular cementum pulled away, but remained attached to the periodontal membrane (Fig. 15). This is only important in that the connection between these two layers may have been of a different character than earlier cemental layers and the cementum attached to the periodontal membrane may have developed subsequent to the operation.

Dr. Orban examined the two histologic sections included in this report. His personal communication and drawing Fig. 16 (modified by the editor), are herewith gratefully included.

"It seems to me that in treatment you were down at a point marked by X and removed part of the surface of the cementum. At this time there might—or must have been a pocket here and no bone. New cementum was laid down up to a point XX —and the distance between X and XX might give the amount of reattachment. The new cementum was artificially torn from the surface part way."

SUMMARY AND CONCLUSION

Evidence of new alveolar bone formation is demonstrated in the case presented through radiographs taken over a period of fifteen years. The histologic specimen

at the end of this period discloses a similar amount of new bone deposition incisally. The periodontal membrane attachment at the base of sulcus, as disclosed by the histologic sections could not have existed at the time of operation. With the increase of new bone incisally and histologic evidence that there now exists an attachment of transseptal fibers on cementum of the canine tooth, it can be concluded that this is a new attachment and at an area where once there was detachment of the periodontal membrane and where a pocket had formally been recorded.

The Relationship of Materia Alba and Dental Plaque to Periodontal Disease

by GRACE C. KIMBALL, PH.D.,* *Cleveland, Ohio*

THE terms *materia alba* and dental plaque refer to accumulations of material on the crowns of teeth. They consist chiefly of microorganisms, food, and cellular debris. The relation of plaques to caries has been studied thoroughly, but the role of such deposits in periodontal disease is largely speculative and based upon clinical observations. This paper consists of a survey of the literature, undertaken as part of a study to explore the possibility that the bacteria in *materia alba* and dental plaque may produce substances irritating to the gingiva, and thus play some part in the initiation of periodontal lesions. That such is the case is an assumption commonly found in the textbooks, with the attention centered chiefly on the proteolytic organisms as opposed to the acid formers, which are more commonly associated with caries.

There is some experimental evidence for an association between proteolytic bacteria and periodontal disorders. In 1947, Berg, Burrill, and Fosdick[1] compared the proteolytic activity of the saliva from 100 patients with periodontal disease, with that from 100 controls. They incubated 15 cc. specimens of saliva plus 100 mg. of casein and measured the protein breakdown products after one hour and after three hours. Analyses were run for hydrolysis of protein by formal titration, and for production of indole, and H_2S. The authors combined these values to derive an index to periodontal disease. The results, with statistical analysis, are shown in Table 1.

	Normal control	*Periodontal disease*
Mean index	11.8	31.51
σ	3.82	13.30
Range	4.47-23.07	14.99-89.55
% within range of standard deviation	68	75
Critical ratio	21.11	

TABLE 1

These results would indicate that the saliva of people with periodontal disease contains microorganisms which are more actively proteolytic than the saliva from people who do not have periodontal disease.

These results suggest that consideration should be given to those organisms, commonly present in the mouth, which play a part at some stage in the breakdown of proteins. By this is meant the organisms which attack any substance in the catabolic phase of the nitrogen cycle, from the com-

Department of Oral Pathology, Western Reserve University.

*This research is supported by a grant from the Colgate-Palmolive-Peet Company.

plete protein down to the final production of ammonia.

Numerous organisms have been reported in saliva and dental plaques. These include protozoa, fungi, spirochaetes, and both aerobic and anaerobic bacteria. Little is known about the metabolic activity of most of these groups outside of the bacteria. This discussion, then, will be limited to those bacteria which constitute the normal flora of the mouth, and those which are strongly proteolytic.

Information concerning the organisms found most often in the mouth is available from the work of Bibby.[2] On the basis of counts made on smears and cultures, he concluded that the cocci predominate in the oral cavity. The ratio between Gram positive and Gram negative cocci varies. In descending order of frequency the oral flora includes also bacilli, fusiform organisms, leptotrichia, actinomyces, vibrios, and spirochaetes.

More data on the relative numbers of different organisms in the mouth is available in Strålfors' excellent monograph in which he summarizes the results of previous workers.[3] Of particular interest is his summary of Bibby's figures. He gives the percentage of the total flora of the mouth which falls into each of six classifications of Gram stain and morphology. The figures are averages from 36 stained smears and are shown in Table 2.

Gram reaction and morphology	*% of total flora*
Gr. pos. cocci	48.6%
Gr. neg. cocci	28.4
Gr. pos. rods	4.4
Gr. neg. rods	6.8
Gr. pos. filaments	4.6
Gr. neg. filaments	7.1

TABLE 2

Strålfors own estimates of the bacterial flora were based on counts made by culture, rather than smears. By plate counts, he found that Streptococci and Neisseria are present in the order of ten[6] organisms per ml., while Lactobacilli number only ten.[3] This agrees in a general way with the results from stained smears as given in Table 2, that the cocci are far more numerous than the rods.

Now the question arises, which of these mouth organisms take part in the breakdown of protein. Fortunately, certain of the steps in this breakdown process are used routinely among the biochemical tests for the classification and identification of organisms. In fact one of these tests, for gelatin liquefaction, depends upon the enzyme gelatinase which also attacks collagen.[5] Therefore this test alone might give a direct indication of those organisms capable of destroying the connective tissue fibers of the periodontal membrane.

Below, the bacteria of the mouth are listed with their pertinent biochemical reactions.[6]

Streptococci—most species do not liquefy gelatin, nor produce indole, or H_2S

Staphylococci—in various series tested, anywhere from 47-100% of strains liquefied gelatin

Neisseria—gelatin not liquefied

Bacilli—i.e., rod forms, would presumably include the following:

- Lactobacillus—gelatin not liquefied
- Bacillus—liquefies gelatin
- B. coli—produces indole, reduces nitrates to nitrites
- A. aerogenes[7]—reduces nitrates—some strains form indole
- Proteus—liquefies gelatin rapidly

Fusobacterium—produces indole and H_2S

Actinomycetes—a few species proteolytic

Vibrios and spirilla—produce NH_3—many species form indole and reduce nitrates—some produce H_2S

The genus *Clostridium* should be mentioned. Although these organisms are found in the mouth of only a minority of the population, they are very actively proteolytic, ranking among the more important of the organisms responsible for putrefaction.

The data concerning the biochemical activity of some of these organisms as determined by Strålfors[8] is even more pertinent, since he tested the chemical reactions (for purposes of identification) of strains of bacteria which had actually been isolated from dental plaques. Of the 12 strains

of Streptococci (11 viridans and 1 salivarius), and 5 strains of Lactobacilli, which were tested, none was active against proteins or their products. Of 6 strains of Staphylococci, 3 liquefied gelatin, and 4 reduced nitrates to nitrites. Of six strains of Gaffkya (*Micrococcus tetragenus*), 3 liquefied gelatin and reduced nitrates. All 4 strains of Sarcina liquefied gelatin. Two strains of Neisseria were not proteolytic. In general, these results agree with the characteristics of the various organisms as reported in Topley and Wilson.[6]

Next, consideration should be given to the chemical substances which are formed during the breakdown of proteins, in order to determine whether this particular metabolic activity of bacteria results in products irritating to the gingival tissues.

There are two different types of protein decomposition. Decay is an aerobic breakdown, producing stable end-products. Putrefaction is an anaerobic breakdown. This incomplete oxidation produces unstable and foul-smelling substances, including mercaptans, amines, fatty acids, indole, and H_2S.[9]

One of the first products resulting from the breakdown of the whole protein molecule is peptone. These peptones are further degraded with the production of many more substances including creatine and creatinine, urea, amino acids, hydrocyanic acid, mercaptans, indole, and several gases, NH_3, CO_2, H_2, and H_2S.[10]

In the above list, the amino acids are the most important in the long chain of protein catabolism. It is interesting that at this point practically all of the bacteria, except most of the Staphylococci and Streptococci, can enter the nitrogen cycle, i.e., can metabolize the amino acids.[11] Porter[12] presents a detailed account of some 10 different mechanisms by which the amino acids may be broken down. These mechanisms may be grouped under 3 main headings, and the end products summarized briefly. The first is deamination, the removal of the amino group from the alpha position in the amino acid. There are 6 different routes by which this may be done. They all yield NH_3, and other products, such as a keto acid, hydroxy acid, or fatty acid, either saturated or unsaturated. The second means by which the amino acid molecule may be broken up is decarboxylation. The carboxyl group is removed as CO_2, leaving an amine. The third reaction is a combination of the first two, deamination and decarboxylation at the same time. This combination of reactions always produces NH_3 and CO_2, while the residue may be a primary alcohol, a hydrocarbon, or a fatty acid.

TOXICITY

Material regarding the toxicity of these various products of proteolysis is rather meager. Peters and Van Slyke[13] discuss one such group which is rather regularly formed in the animal body. This group consists of the toxic aromatic rings, which result from the breakdown of the following amino acids; tryptophane, tyrosine, and phenylalanine. These toxic compounds include indole, skatol, phenol, and paracresol, and their organic acids. They are produced in the body either by the body's own protein catabolism or by putrefactive bacteria in the intestinal tract. These compounds are definitely toxic, and must be detoxified in the liver by conjugation with sulfuric or glycuronic acid before they can be excreted safely in the urine.

Aside from this group of aromatic ring compounds, there is very little definite information available concerning the toxicity of the products of protein metabolism, although it is generally recognized that some of the amines, and the gases, H_2S and NH_3, are toxic when present in sufficient concentration.

Graham[14] investigated the toxicity of the products of proteolysis by injecting pus from periodontal pockets of humans into animal tissues. This, of course, is a nonspecific method which determines the tox-

icity of a mixture of compounds. How much of any observed toxic effect might be caused by substances other than those derived from proteins, can not be determined. The pus was passed through a Seitz filter to remove intact organisms, and injected into cats, guinea pigs, rabbits, and rats. The commonest microscopic finding was fatty degeneration of the liver, and the material proved fatal to 15 out of the 25 animals which were used. The author states that exotoxins have never been demonstrated in such pockets, therefore the toxic substances are probably derived from the tissues. However, there is still the possibility that bacterial endotoxins are present, although quantitatively they would probably be less important than the products of tissue breakdown.

EXTRACELLULAR ENZYMES

There is one other phase of the metabolic activity of bacteria which might be significant in the initiation of periodontal lesions. This is the synthesis of harmful substances by the bacterial cell. This general subject is well treated by Dubos.[15] He divides such substances into three classes: (1) the classic exotoxins, e.g., diphtheria, tetanus, etc., (2) extracellular enzymes which affect the course of an infection, and (3) the endotoxins of the Gram negative bacilli. Classes (1) and (3) can be disregarded for this discussion; Class (1) because infection with tetanus or gas gangrene bacilli are practically unheard of in the periodontal region, and Class (3) because there is practically no information available about the effect of endotoxins on the gingiva.

Under Class (2), extracellular enzymes, Dubos discusses several substances. The first is lecithinase, produced by Clostridium welchii. This enzyme lyses red blood cells, and causes necrosis of other types of cells. There are several organisms which produce extracellular proteinases. Again Clostridium welchii is active, producing collagenase which destroys the reticular framework of muscle. Some strains of hemolytic Streptococci Group A produce an enzyme which destroys the M antigen of other Group A Streptococci. The pathogenic strains of Staphylococci produce coagulase, which causes the coagulation of fibrin. This reaction has two results, which oppose each other, as far as invasiveness of the bacteria is concerned. The fibrin assists in walling off the infection, but it also forms a film on the bacterial cell which protects it from phagocytosis. Another extracellular enzyme is fibrinolysin, also called streptokinase. This enzyme dissolves fibrin clots, thus assisting the invasion of bacteria, and is produced by most of the hemolytic Streptococci of Group A.

An enzyme which has received a great deal of attention recently is hyaluronidase, the so-called "spreading factor." It is produced by Staphylococci, Streptococci, Pneumococci, and some of the Clostridia. Although the actual role of this enzyme as a factor in the invasiveness of organisms is controversial, it will be discussed in somewhat more detail later in this paper, since some experimental work has been done on the action of this enzyme on the periodontal membrane. The last enzymes discussed by Dubos are the Hemolysins and cytolysins. They would not apply to the initiation of periodontal lesions except as an initial trauma might open the way for them to reach the blood cells. Whether the lysis of red cells by hemolysis would interfere with the resistance of the body to infection, is not clearly determined, but the damage to the white cells produced by leucocidins could seriously interfere with the phagocytosis of invading bacteria.

Since hyaluronidase would seem to be a very good possibility as a substance which might aid in the invasion of the oral mucosa by bacteria, it will be considered separately.[16] It is an enzyme which acts on hyaluronic acid, a cementing substance in the body. Chemically, hyaluronic acid is a mucopolysaccharide which is found in the skin, in aqueous humor, synovial fluid, the umbilical cord, and Wharton's jelly. It is hydrolysed by hyaluronidase, which

produces first a lowering of viscosity, then glucuronic acid and glucosamine, and finally acetic acid and ammonia. There is some disagreement as to whether this enzyme increases the invasive power of microorganisms.

There has been one attempt to determine experimentally the importance of this substance as a possible factor in the initial breakdown of the periodontal tissues. This was done by Aisenberg and Aisenberg.[17] There is a theory that the first step in pocket formation is the migration of strands of epithelium downward and between the fibers. These authors reasoned that hyaluronidase produced by bacteria in the gingival sulcus might destroy the ground substance of the connective tissue, and thus permit this penetration by the epithelium. To test this theory, they injected 0.1 cc. of a hyaluronidase solution representing x turbidity units into the interdental papillae at 5-10 day intervals. They present 4 microphotographs which show the resultant infiltration by epithelial strands. However, this experiment leaves two questions unanswered, the effect of hyaluronidase on the surface of unbroken epithelium, and the effect of the injection of an inert substance.

POLYPEPTIDES

There is one more harmful product of bacterial metabolism which should be mentioned, which does not fall into this classification of extracellular enzymes. There has been some interest recently in certain polypeptides derived from animal tissues which are harmful to bacteria. There are reports that certain bacteria also produce polypeptides which are harmful to other bacteria.[18] These are the antibiotics, gramicidin and tyrocidin, both derived from tyrothrycin, which in turn is produced by Bacillus brevis. Whether such polypeptides, at the low concentration produced by microorganisms growing in the mouth, might also be harmful to animal tissues we do not know. However, they would seem to be potentially toxic, since all new concentrated antibiotics must be tested for toxicity before use on humans.

Since there is very little experimental evidence for a relationship between the metabolic activity of bacteria and the initiation of periodontal lesions, a consideration of this problem might be concluded by citing certain clinical and dietary observations which may throw some light on the question. These observations point chiefly to an association between deposits on the teeth and periodontal lesions. From the point of view of this paper, the implication would be that the bacteria in such deposits are an important factor in such lesions.

The general clinical impression is given by Black.[19] He mentions lack of oral hygiene as an important factor in gingivitis. If a sensitive tooth prevents the normal cleansing by mastication on one side of the mouth, or if malformations prevent either natural or artificial cleansing, the affected teeth soon become coated with food debris, and the adjacent gingiva becomes inflamed. Another example of this clinical association is seen in the text of Gottlieb and Orban.[20] The frequent clinical observation of deposits on the teeth along with inflammation of the gingiva had led Gottlieb earlier to coin the term "Schmutz pyorrhea."

Some of the experimental work on diet would also tend to substantiate this idea of a relationship between food deposits and periodontal disease. Mitchell[21] fed a coarse chow diet to hamsters for 98 days, and got only mild periodontal reaction. But a fine, soft, high carbohydrate diet, which would have a greater tendency to accumulate around the teeth, when fed for a similar period, resulted in generalized periodontal disease in practically all of the animals. Burwasser and Hill[22] fed young dogs a diet which varied only in consistency. The controls ate hard biscuits; the experimental animals ate the same biscuits ground and soaked to a mash. After 14 months on this diet, the controls maintained comparatively clean teeth, and healthy gingiva; while the

animals on the soft diet developed plaques on the teeth, and inflamed gingiva. The relative condition of the gingiva was corroborated by histological studies.

On the other hand, observations such as those of Baarregaard,[23] can not be disregarded. These would seem to raise some doubt concerning the relationship of food deposits and periodontal disease. He studied native Eskimos in Greenland, classifying them as primitive or civilized on the basis of their distance from trading posts, with a resultant change in living habits. He found that calcareous deposits and *materia alba* were very common in both groups, yet the primitive Eskimos showed only mild gingivitis, rare periodontitis, and no periodontosis. In contrast, the civilized Eskimos showed 100% gingivitis; periodontitis, with pocket formation and bone loss, was common. However, here too there was no periodontosis. One must conclude therefore, that in this group, at least, food deposits were not the determining factor in the development of periodontal disease.

In conclusion, there appears to be a fairly good theoretical basis for assuming that the bacteria present in *materia alba* and dental plaque might play some part in the initiation of periodontal disease, and that this warrants further experimental investigation.

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The Cementum During Curettage

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BEFORE the loss of the attachment of the periodontal fibers, information on cementum is exhaustive, but after the loss of attachment little is known. A reiteration of the descriptions of the normal is unnecessary, though a review of the literature is profitable, particularly articles by Black,[1] Kronfeld,[2] Orban,[3] and Box.[4]

Black[1] writes: "The cementum is a specialized tissue. Nothing like it exists elsewhere in the animal body. It is in every respect a passive tissue. It does not originate any form of physiological activity. It does not build itself, nor repair injuries to its own tissue. It is laid down on the dentin by the peridental membrane very much as subperiosteal bone is built by the periosteum. It is much like bone, and has in its substance corpuscles very like the bone in its histological content. . . . Cementum differs from bone most widely in the absence of a blood vascular system. In bone every part of the tissue is within the sphere of the circulation of red blood, and without aid from adjacent tissues, is subject to absorption and perfect rebuilding of its own tissues at any time. . . . Cementum has none of these whatever. It has no circulation . . . (and) is therefore dependent upon the peridental membrane for the maintenance of the life of its cement corpuscles. . . .

"When stripped of its peridental membrane *it becomes a dead tissue* (italics the author's), no matter if the pulp of the tooth is alive. The tissue of the cementum has no power of initiating or carrying forward any reparatory process whatever in the absence of the soft tissues around it. . . ."

Gottlieb[5] confirms these observations.

Thus, when the periodontal membrane is detached from the cementum during the progress of periodontal diseases, a dead tissue, the cementum, is in contact with living tissue, the lining of the pathologic crevice. At best, this is biologically undesirable. Dead bone in contact with living tissue can only be an irritant. Were the dead bone true bone, it would sequestrate as a foreign body and exfoliate or resorb. With cementum which has lost its periodontal attachment this cannot occur. Sequestration is impossible, for the cementum is too firmly attached to the vital dentin beneath. Exfoliation is impossible without complete loss of the tooth, and of course the terminal stage of periodontal diseases is complete exfolation of the tooth. Resorption in its usual sense cannot take place, as there is no circulation to provide the cellular elements necessary to this phenomenon.

When a tooth is extracted, periodontal diseases disappear in the area occupied by that tooth. In other words, when the dead tooth surface is removed from contact with living tissues, the recurrent acute and chronic inflammations of clinical periodontitis no longer occur. No other tooth tissue is in contact with these soft tissues but cementum and enamel. Therefore, the source of the irritation causing these inflammations must be something on the surface of the tooth, the tooth surface itself, or both.

Deteriorated enamel surfaces have been discussed in a previous paper.[18]

The irritating factors on the tooth surface are usually identified as organic debris from inspissated saliva and tissue exudate, cellular detritus and food, bacterial growths, calculus, and the putrescent ends of Sharpey's fibers. The removal of these factors and rigid personal hygiene has given beneficial results in many hands since the days of Albucasis and probably before. That this treatment has not been good enough, however, is evidenced by the present widespread use of the various forms

of gingivectomy. Were cases responding satisfactorily to conservative subgingival curettage, we would not have this increasing attention to a more radical approach.

In our own experience, careful removal of the above irritating factors did produce fairly satisfactory results in most cases, but results that were temporary. There were many patients in good health, without too severe periodontal involvement, who cooperated far better than average in home care and in whose mouth the repeated removal of these superficial accumulations seemed to be sufficient treatment for a time. Further experience and closer observation, however, showed that even these were slowly deteriorating in spite of our care. Calculus continued to form, inflammations periodically recurred, and tissue tone was extremely difficult to maintain. Sensitivity at the necks of the teeth and cervical caries plagued us. In the mouths of others who cooperated to the best of their ability, oral hygiene was entirely inadequate, the patient returning at short intervals with sore, bleeding gingivae and filthy mouths.

Both Black[6] and Hartzell[7] made the same observations years ago, and this has been the experience of many operators. One can conclude that removal of the accumulated organic debris on the surface of the cementum is not adequate periodontal treatment.

In addition to the removal of the accumulated organic debris, dentists are usually taught that the surface of the cementum itself must be smoothed and polished, but, not to scratch, or groove, or cut through the cementum, or the healing of the pocket may not take place, and the denuded area will be permanently sensitive.[8,9]

The origin of this belief is difficult to trace. The older practitioners gave it little thought, for they curetted, planed, and polished until the root was hard and smooth, and gave no concern as to what they were removing.[10,11] They expected the tooth to be sensitive, or in other words, they removed the cementum, for the cementum is devoid of any means for the transmission of pain.[11] Others thought they were polishing the surface of the cementum, but indicate from the descriptions of the difficulties they encountered in removing deposits, that they were in reality removing the cementum itself.

Hartzell[8] seems to be the originator of the idea that cementum must be treated with such great care. He wrote that the narrow zone of cement surface between the enamel margin and the gingival pericemental fibers is "normally smooth, and except for accumulations of calculus needs no attention, because it is not so porous as is that portion of the root surface which gives origin to the elastic connective tissue fibers." He also said, "the cementum . . . comprises three easily distinguishable layers, namely, the pitted surface, then a hard layer upon which the pitted layer rests, which separates it from the thick layer of the cementum proper, which . . . contains lacunae, connected by branching canaliculi."

PITTED SURFACE

He believed the pitted surface layer the important one, for it affords a habitat for bacteria. "If, however, we plane the root surface . . . to a depth just sufficient to remove the porous layer, we thereby make it impossible for the bacterial growth to continue," but "cutting deeply into the cementum . . . is hurtful, because it opens the bone cells in the lacunae to infection, for which there is no cure, and too deep cutting is also detrimental because it makes the teeth sensitive to heat and cold."

This seems very confusing. All cementum "gives origin to the elastic (collagenous) tissue fibers," even the acellular cementum in the gingival area. It is not true that it "needs no attention." This area needs more attention than any other, as will be demonstrated in this and subsequent articles. The three layers may be "easily distinguishable" under the microscope, but

they certainly are not "easily distinguishable" to a sharp cutting instrument. The cementum in the gingival area is only as thick as a human scalp hair.[12] Box believes it is beyond the human sense of touch to determine in curettement even when the dentinal-cemental junction is reached, and that because of its thinness, the cementum must of necessity be frequently removed in its entirety. Black[13] and Orban[14] have also recognized this.

For 10 years the author has deliberately removed the cementum in its entirety during the operation of planing and smoothing the root surface in periodontal treatment. Results are definitely superior to any he was able to attain previously, when he was attempting to remove the surface of the cementum.

This is not as radical as it might seem, for it has been the author's observation that most periodontists sooner or later completely remove the cementum and some dentin as well (Fig. 1). One may remove it a little at a time in the form of repeated attempts at subgingival curettage or "scaling"; by repeated prophylaxis and toothbrushing after surgery; or deliberately at the first treatment. It seems more logical to remove this dead tooth surface on the first attempt, removing the foreign body, irritant that it is, in accordance with accepted surgical procedure.

Dr. J. L. E. Banks, with 35 years of subgingival curettage behind him, has shown the author many cases in which he has done just this, many years ago (Fig. 1). He thought he was doing something wrong, but these are the cases in which he has been successful. Those on which he was too careful of the cementum are still actively involved, periodontally, or have been extracted long ago (Fig. 2). Other periodontists in Rochester have done the same. Dr. H. K. Box reports he has also observed this in Toronto.

Probably it is impossible to curet the surface of the tooth root, periodontally involved or not, plane it to smoothness with a *sharp* instrument, and not remove the cementum. Instructions are given to plane to a hard, "velvety" smoothness,[15] like "polished ivory,"[16] yet at the chair the author has been unable to do this without removing the cementum and part of the dentin as well. Neither can this be done on extracted teeth.

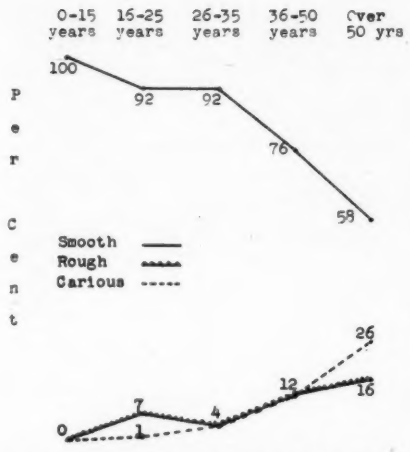
THE STUDY

On the same 500 extracted teeth described in 3 previous articles,[17,18,19] attempts were made to follow the usually described procedure for subgingival curettage: to plane the surface of the root to a hard, glassy smoothness with a sharp scraping instrument.

These teeth were from the mouths of 312 patients as they presented themselves for extraction in the offices of 3 oral surgeons. The teeth from each patient were placed in a separate bottle of zephiran chloride solution, having previously ascertained that months in this solution seems to have no effect on the physical characteristics to be studied. On each bottle the surgeon noted the age, the sex, and the reason for extraction. The statistical subdivisions have been published,[20] each group and sex being represented in sufficient numbers to be significant.

Each tooth was photographed on each of its four surfaces, careful observations of the gross appearance of the cementum in the pathologic crevice area recorded, and measurements to .5 mm accuracy made of the greatest diameter at the cemento-enamel junction and at the bottom of the pathologic crevice as represented by the clinging attached fibers of the periodontal ligament, both bucco-lingually and mesio-distally, for comparison with similar measurements after curettage.

A *sharp* instrument was then repeatedly passed over the cementum in the pocket area, as delineated by the remaining attached fibers of the periodontal ligament, attempting to simulate the usually recommended procedures in subgingival curet-



Graph 1. Visible characteristics of the surface of the cementum in the pathologic crevice area. Average findings on 500 extracted teeth when divided into age groups.

tage. At first the more popular curets and planes, as Gracey's, Towner's, and McCall's, were used, but this was soon found to be too arduous, and being satisfied that the end results are the same, the series was completed with a spoon-shaped Kingsley vulcanite scraper. On the last 93 of these teeth, the entire root was curetted, beyond the clinging fibers of the periodontal ligament, to determine the amount of loss in seemingly normal areas and to see if it were possible to plane cementum to smoothness when it had not lost its soft tissue attachment and been exposed to extraneous influences.

Recordings were made during the planing process as to the character of the surface—whether soft or hard; whether the scrapings were in the form of shavings, flakes, or powder; and whether difficult to remove or comparatively easy.

Measurements of the bucco-lingual and mesio-distal diameters were then made at the same areas as before curettage and compared with the earlier recordings. Photographs were retaken in the same position as before, and the general character of the resulting pattern recorded.

THE CEMENTUM IN THE PATHOLOGIC CREVICE BEFORE CURRETTAGE

The cementum in the pathologic crevice area, from the cementoenamel junction to the remaining clinging fibers of the periodontal ligament, was carefully examined in good light under 2x magnification, to determine any visible roughness or caries, and the findings on each surface recorded. "Roughness" included slight etching, suspected decalcification, definite loss of translucence, and any other change from the smooth, shiny surface one expects on a normal tooth root. *Graph 1* illustrates the findings.

Before 16 years, the cementum on all teeth was still unexposed and appeared normal; between 16 and 25, 74% have the clinging fibers apical to the cementoenamel junction,[17] but only 8% show visible roughening or caries; from 26 to 35, 93%* have exposed cervical cementum,[17] but still only 8% show roughness or caries; after 35, 99% of all teeth have exposed cementum[17] but only 24% have visible roughness or caries between 36 to 50, and 58% after 50 years of age.

In other words, not all exposed cementum becomes visibly deteriorated, though the longer the cementum is denuded, the greater the probability of visible deterioration.

An interesting observation from Graph 1 is the plateau from 26 to 35 years. This plateau has been found in all of our studies, as if the activity of the process causing these deteriorations, slowed or ceased during this interval.

OBSERVATIONS DURING CURETTAGE

As the scraping instrument repeatedly passed over the exposed cementum, no teeth were found whose surface was so hard that the instrument failed to remove visible minute particles. On some, the surface was quite hard and the material removed, a fine powder. At the other extreme, visible caries felt very soft, but as

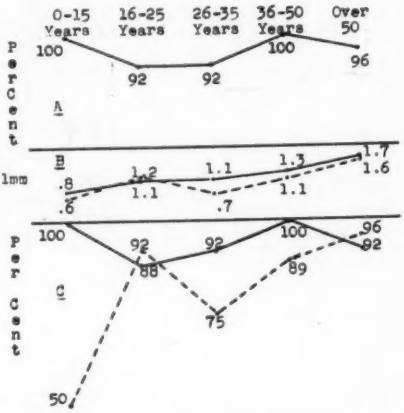
the scraping continued, increasing hardness was felt beneath the caries, until in most the hard, smooth, velvety, "uncuttable" feel so desirable in subgingival curettage was obtained. Between these two extremes of hardness and softness, all degrees of softness were found at the surface, but all followed a definite pattern of progressively increasing hardness as curettage was continued.

For example, on some teeth, particularly from younger patients, two or three strokes of the instrument were sufficient to obtain this glassy hard feeling; on many others, particularly in adult life, the surface was found to be not quite so hard in the beginning, and many strokes of the instrument were required before the desired velvety feeling was obtained; on still others, the surface, even though not visibly deteriorated, was definitely soft, easily planed away, required considerable effort and time to attain our objective, and resulted in considerable loss of tooth structure; on still others, mainly teeth from older patients with very deep pathologic crevices, the surface was so soft that large areas were removed with each stroke and the attainment of our desired hard, almost uncuttable surface, impossible.

Not that the above is universally true, for many young teeth were found with a soft surface and rather deep penetration, and quite a few older teeth with a barely visible loss of structure. All gradations were found at all ages, but in the main they follow a general pattern of increasing softness with increasing age.

Obviously scaling not only removes cementum, but dentin as well. This paper treats only the cementum; the dentin will be discussed in another study.

Observations of the cemento-enamel junction[19] show that the cementum was removed in its entirety during the planing process, by comparing the measurements of the greatest diameter at the cemento-enamel junction both bucco-lingually and mesio-distally, before and after curettage.



Graph 2. Loss of tooth structure at the bottom of the pathologic crevice during curettage. A—Per cent of teeth with a minimum .5 mm loss of structure. B—Overall loss in diameter in millimeters. (Bucco-lingual——; mesio-distal - - - - -.) C—Comparison of per cent of teeth with a minimum .5 mm loss, when subdivided into bucco-lingual (——) and mesio-distol (- - - - -) diameters.

These findings apply equally to this study of the cementum: i.e., the cementum at the cemento-enamel junction was removed during the planing process.

A similar comparison of before and after curettage measurements is shown in *Graph 2*, depicting the loss of tooth structure at the bottom of the pathologic crevice as represented by the clinging attached fibers of the periodontal ligament. This graph represents only 93 of the 500 teeth studied. The first 407 teeth were curetted in such a manner that the instrument did not disturb the clinging attached fibers, duplicating as closely as possible the planing procedure in the mouth. This, of course, did not give a true picture of what would happen to uninvolved cementum—cementum not exposed to the pathologic crevice and with the periodontal ligament still attached. Therefore, the author curetted this last 93 over the entire root to obtain this information, while retaining the first 407 for study of the characteristic pattern formed during curettage—the subject of later studies.

Fig. 1. Male. 64 years old. First treated by Dr. J. L. E. Banks in 1919—32 years ago. Treatment—Conservative (?) Subgingival Curettage. Revisits at about yearly intervals since original treatment.
Note the complete removal of the cementum and part of the dentin, with the hard pink gingivae. The pathologic crevice is almost non-existent in this mouth.

Before 16 years of age, 100% of the teeth had a loss of over .5 mm at what would have been the bottom of the pathologic crevice if there had been one Graph 2A). This is synonymous with findings in the study of the cemento-enamel junction.[19] The average loss at this age was .8 mm bucco-lingually and .6 mm mesio-distally (Graph 2B) and the loss appeared on all teeth bucco-lingually, but on only 50% of the teeth mesio-distally (Graph 2C).

Between the ages of 16 and 25, 92% of the teeth showed an arbitrary minimal .5 mm loss of surface (Graph 2A). This does not mean that the others showed no loss; all showed some, but less than the .5 mm we had set as our minimum accurate measurement. The average loss here (16 to 25) was 1.1 mm bucco-lingually and 1.2 mm mesiodistally, the bucco-lingual being affected in 88% of cases and the mesio-distal in 92% (Graph 2B & C). This is the only time that the mesio-distal exceeds the bucco-lingual in either amount of loss or frequency of incidence, and one would be tempted to consider it a chance result if similar studies of the other 407 teeth had not shown the same trend in this age grouping.

As time goes on, and the bottom of the pathologic crevice recedes,[17] the number of teeth with lost substance at the bottom of the crevice is still at 92% between 26 and 35, rising to 100% from 36-50, and dropping to 96% past 50 (Graph 2A). The amount of loss decreases on the mesio-distal between 26 to 35, and then both bucco-lingual and mesio-distal losses slowly increase in amount through life (Graph 2B). At the same time the mesio-distal incidence of loss returns to its usual position of less than the bucco-lingual loss, and then gradually occurs more frequently until after 50, when it is again more common than the bucco-lingual.

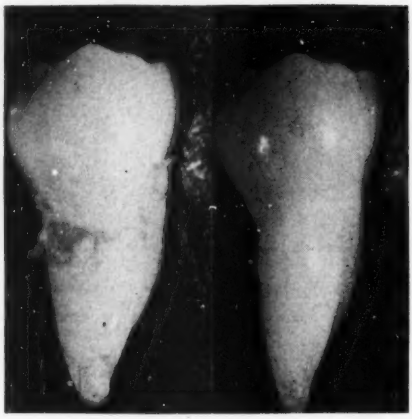
In other words, on most teeth at all ages, there was a minimal .5 mm loss of substance just below the bottom of the pathologic crevice. On the teeth where the loss was less than .5 mm, there was a visible loss during the planing process, probably in excess of the hair-like thinness of cementum. In addition, not shown on the Graph, each tooth on which the entire root was curetted showed sufficient loss to give the impression that the cementum had been removed over the whole root surface, though no attempt was made to measure this accurately (Fig. 3).

A .5 mm loss, either bucco-lingually or mesio-distally, would average 2.5 mm on each surface, which is 5 times the greatest estimated thickness of the cementum in the area of the cemento-enamel junction, and greater than the estimated thickness in the apical area.[20]

Characteristically, the loss is always greater at the cemento-enamel junction and shallowest at the bottom of the pathologic crevice. The area of cementum between these two would thus be removed at least as much as at the bottom of the crevice.

From this one can conclude that the cementum in the pathologic crevice area is removed in its entirety during the planing of the root surface during curettage and it is probably impossible to curet the cementum to a smooth, hard, "velvety" surface without removing it in its entirety.

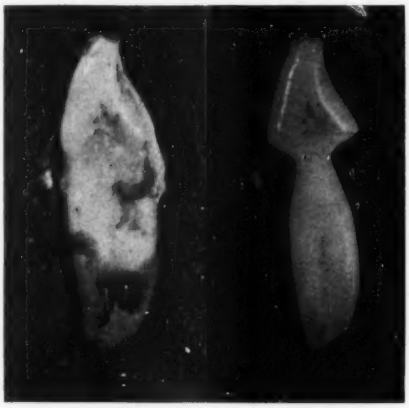
Fig. 2. Female. 35 years old. History of many years of Conservative Subgingival Curettage. Obviously unsuccessful.



Before Curettage. After Curettage.

DISCUSSION

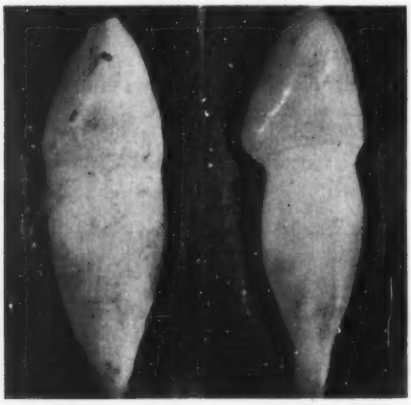
Even if the cementum could be curetted with a sharp instrument to the much recommended hard, glassy surface, without removing it; even if non-viable cementum were biologically inert, instead of the permeable[21] foreign body that it is: still, exposed cementum would be undesirable. To quote Cahn[22]: "Cementum is quite analogous to bone and like bone, should never remain bare. Just as bone must be covered with periosteum, cementum must be covered with peridental membrane. When it is not covered, cemental necrosis takes place. Caries of cementum is found most often where there are periodontal le-



Before Curettage After Curettage

Fig. 3. The Removal of the Cementum in its entirety as the result of curetting the entire root surface with a sharp instrument until the smooth, hard, "velvety" feel so desirable in subgingival curettage is obtained.

Before Curettage. After Curettage.



sions." Evidence of this was offered above in the observations of the increasing incidence of visible deteriorations on the cementum in the pathologic crevice with increasing age.

Chief among the arguments of some, cautioning care in the treatment of the cemental surface, is that if the cementum is removed, reattachment of the soft tissues will not take place. One would hesi-

tate to say that reattachment cannot take place in the face of the enthusiasm of several observers. The author would be even more hesitant to plan treatment with the expectation of reattachment in the light of present experience. Most periodontists have had cases where reattachment seems to have occurred, but continued observation finds these successes temporary, with the pathologic crevice recurring, and the usual symptoms of periodontal diseases again making themselves manifest. The author looks upon a disappearance of the pathologic crevice following treatment, without a corresponding lowering of the height of the gingival crest, with suspicion and distrust. Even the advocates of reattachment do not claim it for the majority of cases, and all seem to agree that reattachment is an ideal for the future, not something to expect in every case. Should one continue to expect reattachment, however, Box[25] believes that exposed dentin presents a suitable medium, and more recently Linghorne and O'Connell have demonstrated this experimentally.[26]

CONCLUSIONS

Cementum which has lost its periodontal ligament attachment is a dead tissue. In contact with living tissue it becomes a foreign body and an irritant which cannot be sequestrated or exfoliated.

Removal of the organic debris from the surface of the cementum is not adequate periodontal treatment.

Planing the outer third of the cementum, as taught by Hartzell and others, is irrational, undesirable, and impossible.

Ten years of deliberate removal of the exposed cementum during subgingival curettage by the author has resulted in definitely better results than those obtained when care was exercised to plane the outer surface only.

A study of 500 extracted teeth demonstrates the impossibility of curetting the root surface to a hard, glassy smoothness without removing the cementum in its entirety.

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(Continued on page 191)

Case Report

Chromic Acid Burn

by MARVIN M. SUGARMAN, D.D.S., *Atlanta, Georgia*

ALTHOUGH there are many improved drugs available, the empirical use of chromic acid has persisted for many years. Dobbs[1] pointed out that the judicious use of chromic acid must be based on an understanding of the nature of the biochemical effects and properties of the drug. Chromic acid, $Cr(OH)_2O_2$, is a powerful oxidizing agent sometimes used in medicine and dentistry as a caustic. It destroys tissue cells and bacteria with which it comes in contact. The therapeutic property of chromic acid depends upon the oxidizing action, its high acidity and its precipitation action on proteins.

Dobbs[1] carried out experiments by painting one half of the labial surface of extracted teeth five times with 10% solution of chromic acid. The painted areas showed softened and pitted enamel.

Glickman and Johannesen[2] reported a study of the effect of 6% chromic acid on the gingiva of the albino rat. They showed a single application produced an injurious effect on healthy gingiva. Gingival repair that took place after the use of chromic acid resulted in an incomplete restoration to the pre-existing level.

Crater formation and the loss of interdental papillae following the use of chromic acid over a long period of time has been shown. In addition chromic acid is such a powerful caustic that it may cause burns and ulcerations.

Formerly, the use of 5 to 10 per cent solution of chromic acid was acceptable in the treatment of Necrotizing Ulcerative Gingivitis. Today the Accepted Dental Remedies[3] of the American Dental Association lists the use of chromic acid as contraindicated because of the better available drugs.

CASE REPORT

A white man, aged 60, complained of soreness in the oral cavity of a week's duration. He stated that he had been treated for "trench mouth" with chromic acid daily for the four previous days. On this day he had penicillin from his physician for his mouth infection. Clinical examination revealed very edematous lips and adenopathy of the submaxillary glands. The temperature was slightly elevated and the patient appeared ill. There were acute ulcerations in the labial and buccal folds.

The gingivae were pink, well-formed and showed no signs of ulceromembranous sloughing. There was no history of metallic taste or increased salivation. The labial and buccal folds were markedly ulcerated. These large ulcerations appeared to be a result of the continued use of the chromic acid. The buccal and labial ulcerated areas were so large that an allergic reaction was suspected. The patient was patch-tested using the technique and strength suggested by Sulberger and Baer.[4] The results showed no allergy to chromic acid.

TREATMENT

Treatment of the burns consisted of the use of warm solutions of Na, Co3 hourly as a mouth wash. Topical application of 2% aqueous solution of Methelyn Blue was used daily. Healing took place uneventfully but slowly. The ulcers healed from the

Figure 1

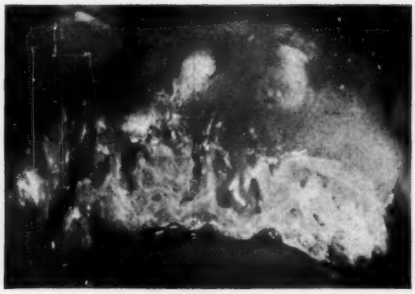

Figure 3

Figure 2

Figure 4

The above four figures show the acute reaction of tissue to chromic acid. In Fig. 1 observe the healthy intact gingiva. In Figs. 2, 3, and 4, note the large ulcerations, desquamations and necrosis of the mucous membrane in the buccal and labial folds.

borders toward the center and required eight days.

SUMMARY

A discussion of the use of chromic acid and a case report of a chromic acid burn has been presented. The disadvantages of this drug as a therapeutic agent has been pointed out. With the scope of new drugs at our disposal, the use of chromic acid in dentistry is unnecessary, contraindicated, and should be discouraged.

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Periodontosis

Review of the Literature and Case Report

by GEORGE J. WHINSTON,* D.D.S., *New York, New York*

PERIODONTOSIS, as defined by the 1949 Nomenclature Committee of the American Academy of Periodontology,[1] is a general term used to include a degenerative non-inflammatory destruction of the periodontium originating in one or more of the periodontal structures. It is characterized by migration and loosening of the teeth in the presence or absence of secondary epithelial proliferation and pocket formation or secondary gingival disease. Because periodontosis is so significant in the current evaluation of perio-disease.

Diagnosis—Periodontosis usually involves all the teeth of both jaws. In some cases, however, individual teeth or groups of teeth may be affected more than others. The most common groups affected are the first molars and the incisors. Goldman[2] calls attention to the fact that migration of the maxillary incisors is an early characteristic symptom and is accompanied by loosening of the teeth. The loosening and wandering may be the only clinical symptoms. This is unlike periodontitis, where such signs usually do not appear in the early stages of the condition. The migration is a slow process and is most often restricted to the single-rooted teeth because they offer less resistance to the occlusal forces than the multi-rooted teeth. In the early stages of periodontosis, the author explains, no inflammation of the gingivae or flow of pus is noticed. Only in the later stages does pocket formation occur. When pocket formation does take place, usually on the mesial or distal surfaces, a marginal gingivitis may result, due to the lodging of food debris. Percussion of an affected tooth may give rise to a dull sound because of the presence of loose edematous connective tissue around the root. Although calculus is usually found around the neck of the tooth, it is not responsible for the pocket formation. However, it does cause an inflammatory gingival condition which may be found in the later stages. Pain is not a primary sign of periodontosis. A painful sensation, though, may result from sudden changes in temperature or irritation from acids, sugar, and other substances. Pain can also occur from excessive mobility of the teeth during mastication. The X-Ray is an important diagnostic aid, for the extent of bone involvement is best determined by radiographic examination. Widening of the periodontal membrane space is characteristic. In most cases the lamina dura is missing or hazy. The alveolar bone may show a general haziness, with indistinct trabeculae.

In his studies to determine the etiology of periodontal disease, Karshan[3] found several characteristic signs present in the group of cases diagnosed as periodontosis. As a rule, the gingivae appeared pink and stippled, with a slight erythema of the interdental papillae in several areas. Little or no calculus was present. There was migration of the maxillary and mandibular incisors. X-Ray examination revealed loss of alveolar bone which usually conformed to a definite pattern. The loss of bone most often extended from the mesial surfaces of the second molars to the distal surfaces of the second premolars, as well as around the incisor teeth. In many areas there was a thinning of the alveolar crest and a widening of the periodontal membrane. More alveolar bone was present about the premolar teeth in both jaws, particularly in the mandible. It was noted that local factors were either absent or not prominent and apparently did not ac-

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*Captain, Dental Corps Reserve. Recently relieved from extended active duty.

count for the severe loss of alveolar bone.

Miller[4] states that young adolescents with periodontosis seek treatment because one or more molars have become mobile or an incisor has migrated. In most cases there is no complaint of pain. X-Ray examination reveals an existing alveolar bone resorption which is advanced. The prognosis at this stage may be hopeless.

Etiology—Although in most cases periodontosis cannot be associated with any obvious disease process, Goldman[2] says that the condition may at times be accompanied by systemic diseases. Some of these, he suggests, may be diabetes, undulant fever, nephritic conditions, tuberculosis, endocrine disturbances, scleroderma, and nutritional disturbances. It is considered probable that the periodontium is affected through another process initiated by the disease, rather than by the disease itself.

On the other hand, though, Kerr[5] believes that periodontosis may actually be a manifestation of a systemic disturbance, particularly of a collagen disease. Several conditions such as periarteritis nodosa, lupus erythematosis, and dermatomyocitis, are classified as collagen diseases. They are characterized by a degeneration of collagen fibers. Since the pathological picture in the collagen diseases resembles the degenerative process found in the periodontal membrane, the above investigator feels that periodontosis may be a manifestation of collagen disease.

Karshan,[3] in his investigation of the causes of periodontal diseases, referred to previously, included a group of 77 cases diagnosed as periodontosis. The mean age of this group was 27.3 years. A group of 51 patients free from periodontal disease was used as the control. The results of the various tests performed to determine the etiology were revealing. Although the experimental groups (including the 77 cases of periodontosis) and the control group both deviated slightly from the normal, no significant differences were found between the two groups for the following blood constituents: total calcium, inorganic phosphate, sodium chloride, total protein, total cholesterol, non-protein nitrogen, urea, uric acid, carbon dioxide capacity, hemoglobin, Vitamin A, and carotene. However, there were more deviations from the normal in the experimental groups than in the control group for the following additional constituents of the blood: vitamin C, glucose, globulin, albumin, calcium ion, alkaline phosphatase, red blood cell sedimentation rate, red blood cell count, white blood cell count, and neutrophils. The plasma vitamin C was low in a large percentage of periodontosis cases in spite of an adequate intake of this vitamin. This could indicate that there was some interference in the utilization of the vitamin. Although a difference was found between the control and the periodontosis group for the ketosteroid content of urine, it is as yet uncertain whether adrenocortical dysfunction is related to periodontosis. (The determination of the 17-ketosteroid output is essentially an index of adrenocortical activity.) The Kline test was negative in all cases and no difference was found between the two basic groups for the basal metabolic rate. Physical examinations and case histories in 44 of the 77 periodontosis patients gave essentially negative findings in 17 of them. Several apparently unrelated conditions were revealed in the remainder of the group. The investigator concluded that from the available data it did not seem as though a common systemic condition acts as a predisposing factor in periodontosis.

Pathology—In the development of periodontosis, Orban and Weinmann[6] pointed out three stages pathologically. They are summarized as follows:

> The first stage is characterized by a degeneration of the principal fibers of the periodontal membrane. Immediately following this change there is probably a cessation of cementum formation. Concurrently, the alveolar bone shows signs of resorption due mainly to an increase in tissue pressure. This latter condition results from a proliferation of capillaries and the accumulation of edema in the loose fibrous tissue replacing the principle fibers.

The second stage is characterized by the rapid proliferation of the epithelial attachment along the affected cementum. The first signs of inflammation may be revealed along with the epithelial proliferation, probably due to the penetration of toxins from the gingival sulcus. Clinically, the second stage cannot be differentiated from the first stage. Both stages are of short duration. However, there is the possibility of repair in the first stage, whereas the second stage must progress to the last stage.

The third stage starts with the development of deep pockets, which are caused by the degeneration of the epithelial tissue at the base of the gingival sulcus. Clinically, the third stage is most frequently encountered.

The microscopic studies of the cases reported by Goldman[2] reveal additional pathological changes. The fatty marrow of normal alveolar bone is replaced by loose, fibrous connective tissue with many dilated blood vessels. There is a distinct increase of the capillaries in the marrow spaces. In the later stages, the periodontal membrane may be widened as a result of the resorption of the alveolar wall or the movement of the tooth. In some cases studied the alterations in the bone were most prominent. The osteoclasts were multinucleated and numerous. It is pointed out that the apical proliferation of the epithelial attachment occurs when the principal fibers lose their attachment to the cementum. The principal fibers concerned are the free gingival, transseptal, and the alveolar crest fibers. Once the attachment of the transseptal and the alveolar crest fibers has been lost, due to degeneration, the gingival fibers relax and allow proliferation along the cementum to occur. This is responsible for the pocket formation ultimately occurring in Orban and Weinmann's third stage.

Pathologic wandering, also described by Goldman,[2] is associated with changes in the periodontal membrane. The destruction of the principal fibers allows the tooth to move by a contraction of the remaining intact attachment of the other side of the tooth. The tooth thus tends to move in the direction opposite that where the periodontal membrane is involved. Adding to the contraction of the intact fibers is the simultaneous push force resulting from the edema on the affected side.

From the above descriptions, it is apparent that the earliest pathologic changes in periodontosis occur in the periodontal membrane. This is followed closely by the alveolar bone.

Treatment—In most cases, the treatment of periodontosis is to no avail. The progress of the condition usually continues, with increased mobility resulting. Goldman[2], however, offers several suggestions for the attempted checking of the condition. He believes that every case should be thoroughly investigated. A complete physical examination should be carried out, as well as all indicated laboratory studies. If any of the findings are positive, treatment is instituted accordingly. If no abnormalities are found, it is advisable to prescribe a well balanced diet. This should be supplemented by vitamin therapy. Good health and environment are of most importance.

The case should be treated by the removal of existing symptoms. By so doing, the progress of the disease may be cut down considerably, perhaps disappearing altogether. Such treatment should include the elimination of periodontal pockets, preferably by surgical gingivectomy. The occlusion should be equilibrated so that the teeth will be in harmony with one another. Stress on the individual tooth should be decreased by reducing the occlusal surface. This may be done by cutting down the bucco-lingual width. To help prevent occlusal disharmony brought about by the migration of teeth, splints of either the fixed or removable type may be indicated. Good oral hygiene must be maintained to overcome secondary inflammation.

It is understood, of course, that such treatment is attempted only if the condition has not reached the advanced late stage. In such cases, it is usually advisable to extract all involved teeth due to the discomfort to the patient and the hopeless prognosis.

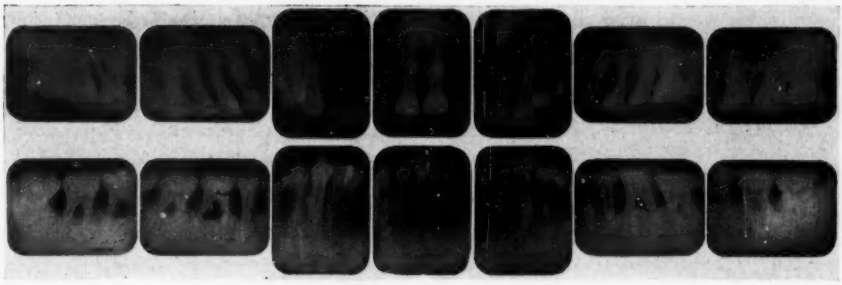

Fig. 1. Diffuse advanced alveolar bone resorption present. There is bifurcation involvement of the mandibular first and second molars and trifurcation involvement of the maxillary first and second molars. Lamina dura is not present in molar and premolar regions. Notice widening of periodontal membrane space in maxillary molar and incisor regions.

CASE REPORT

A 29 year old male Negro reported to the dental clinic on Dec. 4, 1951. His chief complaint was a feeling of "numbness" in the gingival tissue of the left maxillary premolar and molar region. Occasionally, a dull pain was present in the area.

History of Present Condition—The patient first began to have the feeling of numbness in September, 1951. The sensation gradually became more marked. About the middle of October, 1951, the patient began to have a dull pain in the same region. The symptoms of numbness and intermittent dull pain continued up to the time the patient presented himself at the dental clinic.

Examination of Oral Cavity—Clinical examination revealed a normal healthy mucosal lining. Caries was not evident in any of the teeth. Aside from a mild marginal and papillary gingivitis in some areas, the gingival tissue was pink in color, with occasional dark gray pigmented areas. The distribution of the gingivae was essentially normal. There was no recession. There was a slight to moderate degree of subgingival calculus, usually limited to a narrow ledge around the neck of the tooth. Upon palpation of the gingival tissue, a soft spongy, edematous consistency was noted. In addition, upon pressure, a white caseous material, resembling pus and soft debris, was expressed at the margin of the gingiva of all teeth. Bleeding of the gingivae did not occur spontaneously and there was no previous history of persistent bleeding. Exploration revealed exceedingly deep pockets around all the teeth. These pockets were found on all surfaces. The maxillary right central incisor was elongated and in labial version to the adjacent teeth. This was considered to be the result of migration. All teeth had considerable mobility. Several teeth could be depressed in the sockets, particularly the mandibular right and left first molars and the maxillary left first premolar. The occlusion was generally good, with no significant areas of traumatic occlusion in central relation or the lateral and protrusive movements.

Radiographic Examination—(Fig. 1)—The X-Rays revealed all the teeth to be free of caries. Calculus could be clearly demonstrated around the necks of certain teeth. A diffuse advanced alveolar bone resorption existed. About ½ to ⅓ the length of alveolar bone remained in the incisor region. There was bifurcation involvement of the mandibular right and left first and second molars. There was trifurcation involvement of the maxillary first and second molars on both sides. The bone resorption had progressed to the apical regions of the maxillary left first premolar and the maxillary right first molar. There was a widening of the periodontal mem-

brane space around most of the teeth, particularly the maxillary and mandibular molars on both sides and the maxillary incisors. The lamina dura was missing in all premolar and molar regions. It was indistinct in the canine and incisor regions. The least amount of bone loss was demonstrated in the right premolar regions. In general, a horizontal type of bone resorption was occurring.

Impressions and Diagnosis—The periodontal condition was of such a severe nature that the existing local factors did not appear to be the basic cause. The advanced alveolar bone resorption was out of proportion to whatever local irritating conditions were present in the mouth, mainly calculus and possible food impaction. The condition in the mouth could very well have been a manifestation of an underlying systemic disorder. Considering such a possibility, it was decided to have the patient undergo a medical check-up. Since the characteristics of the bone loss were very similar to those found in hyperparathyroidism, special emphasis was placed on studies to detect a hyperparathyroid condition or a disturbance in calcium and phosphate metabolism.

It is pointed out by Miller[4] that in hyperparathyroidism there is hyperfunction of the parathyroid glands resulting from an adenoma. The blood phosphorus is diminished while the blood calcium is increased. Excessive excretions of calcium and phosphorus occur in the urine, resulting in the secondary formation of fibrosed cysts in the bone. However, it is brought to our attention that cyst formation is not always present in the bones and normal blood levels of calcium and phosphorus may be found in cases considered to have hyperparathyroidism. Under such circumstances, renal failure (lithiasis) may be the only finding. Consequently, in performing tests to rule out the possibility of hyperparathyroidism, the foregoing facts should be kept in mind for correct interpretation.

From the clinical appearance and radiographic studies, the periodontal disorder confronting us was classified as periodontosis, advanced stage.

Physical Examination—The patient did not appear ill. He was of better than average intelligence. History revealed that the patient had headaches for the past ten years, occurring practically every day. For the past five years the patient recalls having a tired, fatigued feeling. This has become more noticeable since September, 1951, when the patient returned to the United States after eleven months in Korea. The patient was frequently constipated.

Head and neck: normal, except for palpable thyroid gland which was not enlarged. Eyes, ears, nose, and throat: normal. Chest: normal. Lungs: clear to percussion and auscultation. Cardio-vascular: heart not enlarged, rhythm regular, no murmurs. Abdomen: flat, no rigidity or tenderness. Genitalia: normal adult male. Rectum: no hemorrhoids, no masses, stool brown. Prostate: normal size and consistency. Back: no abnormalities. Extremities: no abnormalities. Neurological: no abnormalities. Lymphatics: no adenopathy. Skin: no abnormalities.

Laboratory Studies—Note: Since tests were performed on several occasions over a four week period, more than one value may appear for some tests.

Urinalysis

Color—Appearance: yellow, clear
Specific gravity: 1.030; 1.029
(Normal: 1.016-1.023)
Albumin (Qualitative): Negative
Sugar (Qualitative): Negative

Hematology

White Blood Cells: 7,200; 8,100; 10,100
Red Blood Cells: 4,800,000; 4,100,000
Differential Count:
Neutrophils: 47%; 70%
Lymphocytes: 48%; 28%
Monocytes: 2%
Eosinophils: 3%; 2%
Hemoglobin: 14 gm.; 13.5 gm.
Sedimentation Rate: 1 mm/hour; 2 mm./hour
(Normal: Very slight or no sedimentation in 1 hr.)

Blood Chemistry

Calcium: 10.9 mg./100 cc. blood; 10.8 mg./100 cc. blood
(Normal: 9-11 mg./100 cc. blood)
Phosphorus: 2.8 mg./100 cc. blood
(Normal: 3-4 mg./100 cc. blood)

Serology

Routine Kahn Test—Negative

Feces

No ova or parasites were seen.

Sulkowitch Test

This test is used to determine whether there is an elevation in the calcium content of the urine above normal. A relative increase of calcium is detected by a precipitation occurring when reagents are added to the urine. This is a convenient method of demonstrating an increased amount of calcium in the blood and urine, for if there is such an elevation, it will readily reveal itself. A zero reading would indicate a hypocalciuria and hypocalcemia, while a 3 or 4 plus would represent a hypercalciuria and hypercalcemia. Three Sulkowitch tests gave a reading of 2-plus.

Total Urinary Calcium Following Low Calcium Diet

The patient was put on a diet containing less than 100 mg. of calcium per day. The total calcium excretion in a 24 hour urine specimen was determined on the seventh day. The result was 121 mg. of calcium excreted in a 24 hour period. Since the normal range is 50 to 150 mg. per 24 hours, the findings were not abnormal. If there existed a definite hyperparathyroid condition, an increased value for calcium excretion would have been obtained even after a low calcium diet.

Radiographic Studies (Systemic)—Excretory studies of the urinary tract showed no abnormality of the upper or lower collecting systems.

Examination of the chest revealed no pulmonary infiltration. The thoracic cage presented no abnormality in texture. As determined by height and weight charts, the heart was enlarged approximately 10%. This enlargement was predominantly of the left ventricle. A skeletal survey including films of the skull, forearms, lower legs, knees, and pelvis showed no alteration in bone density, structures, or development.

Conclusions—On the basis of the physical examination, laboratory studies, and radiographic reports, it was the belief of the physicians of the internal medicine section of the hospital, who conducted this phase of the work-up, that there was no evidence of a disturbance in calcium or phosphate metabolism or hyperparathyroidism. In addition, the data obtained did not at the time suggest any other possible systemic disturbance which could give rise to the periodontosis.

Shortly after the conclusion of the tests, the maxillary left first premolar was extracted because it became extremely tender to percussion and palpation. On the day following the extraction, the patient stated that the peculiar numbness he had experienced in that region was considerably less. It was assumed that the involvement of the bone structure in the apical region of the tooth plus its marked mobility had produced, in part, the original symptoms.

The remaining teeth were subsequently extracted because of the extreme mobility present and the belief that from then on acute episodes would continue to occur as individual teeth became symptomatic. The degenerative process was of such a nature that it would not cease.

Comments—This case of periodontosis is of considerable interest because it strikingly presents many of the classical characteristics described in the literature. Outstanding among these is the fact that it could not be associated with any single systemic condition. However, the possibility remains that the answer lies within the intricate mechanisms of the body. This factor is significant and deserves further investigation.

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Announcements

Winners of the Student Essay Contest of the
American Academy of Periodontology, 1951-52.

The American Academy of Periodontology is sponsoring an annual essay contest designed to promote interest in periodontology among undergraduate students. The contest is in two general parts:

1. *Local Contest* in each school. Each year the American Academy of Periodontology wishes to award a year's subscription to the JOURNAL OF PERIODONTOLOGY to the undergraduate student who writes the best paper on periodontia in each college. Each faculty of each college can make its own rules and regulations and select the winning paper. The winner's name should be sent to the Editor of the JOURNAL before May 15 of each year. This year the winners are:

University of Buffalo School of Dentistry, John J. Joyce
Creighton University School of Dentistry, Clement J. Stachowicz
Harvard School of Dental Medicine, James P. Ryan
Indiana University School of Dentistry, W. L. Ward
University of Nebraska College of Dentistry, G. D. Kloeffler
New York University College of Dentistry, Lawrence A. Weinberg
Northwestern University Dental School, Kenomi Kanazawa
University of Oregon Dental School, Paul W. Kunkel and Lester A. White
University of Pittsburgh School of Dentistry, Wm. R. Grubler
Temple University School of Dentistry, Herbert Katz
University of Washington School of Dentistry, Frank C. Burrell

2. *National Contest.* Each year one paper from each school can be submitted to the National Contest for the three best papers written by undergraduate students. The winners of the contest this year are:

1st prize: "Hormones and the Periodontium" by Dr. W. H. Christianson, University of Washington School of Dentistry, Seattle, Washington.

2nd prize: "A Visualized Technique for Equilibration of Atypical Occlusal Relationships" by Dr. Lawrence A. Weinberg, New York University College of Dentistry, New York, New York.

3rd prize: "A Consideration of Bone Changes in Occlusal Trauma" by Dr. Herbert Katz, Temple University School of Dentistry, Philadelphia, Pa.

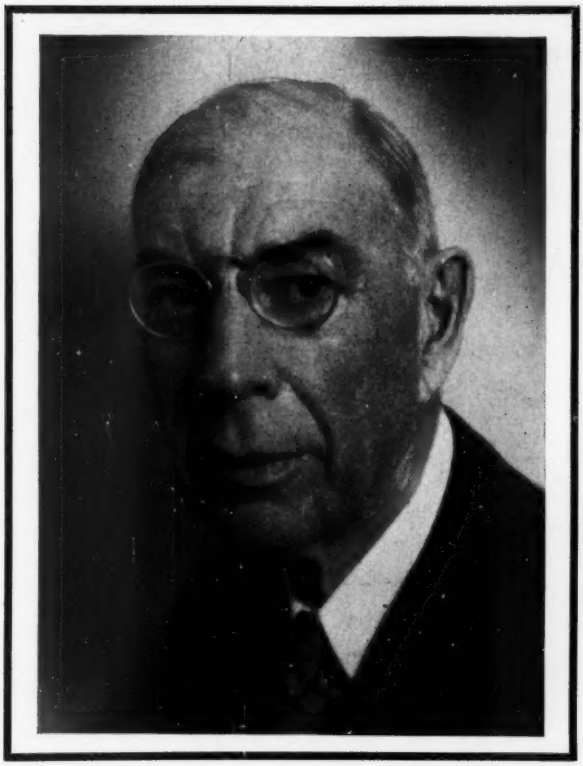
In addition, the Committee voted to award honorable mention to:

Dr. William R. Grubler for his excellent review of the literature on "Radiographic Appearance of the Alveolar Bone and Associated Supporting Structures in Periodontal Disease"

and to

Dr. Paul W. Kunkel and Dr. Lester A. White because their paper reported some original research.

CONGRATULATIONS TO ALL OF THE WINNERS!



AUSTIN FLINT JAMES

August 21, 1870 — April 13, 1952

Austin Flint James was born at Bentley, Illinois, the first child of Joseph A. James, M.D., and Elizabeth Allison. He was named by his father in honor of the famous surgeon of that day, Austin Flint, whom he greatly admired. After a period of attendance at Carthage College, Austin Flint James entered the Chicago College of Dental Surgery (now Loyola) and was graduated with the degree Doctor of Dental Surgery in 1892. As an undergraduate he became a member of Beta Chapter of Delta Sigma Delta and continued the fraternal association actively throughout his life.

He entered practice in 1892 in Oak Park, Illinois, a suburb of Chicago, and continued there for ten years, moving then in 1902 into downtown Chicago. In 1912 a need for better understanding of the biologic factors involved in dentistry led him to become one of the founders of the Chicago Dental Research Club, which, with occasional reinforcements and replacements, carried on for thirty-eight years. The most widely known activity of this group was the nutritional experimental investigation at the Mooseheart Home in Illinois, which was documented in the publication of "Diet and Dental Health" by Hanke in the early '30s.

In 1914, at Washington, D. C., in the first annual meeting of The American Academy of Periodontology, Austin Flint James was elected President. This was recognition by the founders, who felt the first president should not come from their own small group, of his intense interest in prevention of disease and the maintenance of health of the investing structures of the teeth. Because the first year of the Academy was largely formative, he was re-elected President in 1915 and subsequently throughout his life The Academy of Periodontology was the center of all his thinking, in a life which appeared to be entirely wrapped up in the profession of dentistry. In later years he was awarded a fellowship in the Academy, and with the formation of The American Board of Periodontology became a Diplomate and later served upon the Board.

After fifty-three years of active practice an accident and gradually increasing ill health forced his retirement to California. Briefly he attempted part-time practice in California in his beloved field of periodontia, but failing vision shortly terminated this. He had held a license in the State of California since 1926 and had intended to retire there five years before ill health forced it upon him. The loss of his wife, Jennette Irvin, in 1939, however, altered the zest he had previously shown for the new venture in California and he determined to remain in harness in his environment of many years. His death on Easter Sunday of 1952 released him from an existence which had been plagued by discomfort and pain for the past few years but left a void in the lives of those who knew him.

ANNOUNCEMENT

DENTAL MEDICINE SEMINAR

9th annual seminar for the study and practice of dental medicine will be held in the Desert Inn, Palm Springs, California, October 26-31, 1952.

This is the 9th annual seminar to be held and as in the past has an excellent program. Speakers include:

Dr. Hermann Becks	Dr. Ralph Reynolds
Dr. Wm. Rettberg	Dr. Harry Sicher
Dr. Wm. Wainwright	Dr. Herbert Evans

Anyone interested should write Miss Marion G. Lewis, Executive Secretary, Room 200, Hooper Foundation, U. C. Medical Center, San Francisco 22, California.

FELLOW IN ORAL PATHOLOGY TO BE APPOINTED

A new fellow in Dental and Oral Pathology will be appointed some time this fall. This fellowship is sponsored by the American Dental Association through its Council on Dental Museum and Registry.

The award is for $3600.00 annually and the training is given at the Armed Forces Institute of Pathology in Washington, D. C., and is fully creditable toward board certification.

Those interested should contact Col. Joseph L. Bernier, DC, USA, Secretary-Pathologist at the Armed Forces Institute of Pathology, Washington, D. C., as soon as possible.

ST. LOUIS, MISSOURI

PROGRAM FOR ANNUAL MEETING

The American Academy of Periodontology

Statler Hotel

St. Louis, September 4-6

Thursday—September 4

9:00-11:00 a.m.	Business meeting.
11:00-12:00 a.m.	Prevalence and Assessment of Gingival Disease. —Dr. Isaac Schour, Chicago Illinois
2:00- 3:00 p.m.	The Etiology of Periodontal Disease. —Dr. Paul E. Boyle, Philadelphia, Pa.
3:00- 4:00 p.m.	Present Concepts of the Treatment of Periodontal Disease. —Dr. Henry Goldman, Boston, Mass.
4:00- 5:00 p.m.	The Physiology of the Masticatory Mechanism. —Dr. Bernard Jankelson, Seattle, Wash.

Friday—September 5

9:00-11:00 a.m.	The Correction of Occlusal Disharmonies Based on Functional Analysis of Casts. —Dr. Arne G. Lauritzen, Seattle, Wash.
11:00-11:30 a.m.	Discussion of the Problem of the Correction of Traumatic Occlusion —Dr. Bernard Jankelson
11:30-12:00 a.m.	Summary Discussion of the Techniques of Occlusal Equilibration. —Dr. Clyde H. Schuyler, New York, New York
12:30- 2:00 p.m.	Round Table Luncheon
2:15- 3:00 p.m.	Oral Manifestations of Systemic Disease. —Dr. Lester W. Burket, Philadelphia, Pa.
3:00- 3:20 p.m.	A Study of the Interdental Alveolar Bone Crest. —Dr. Beryl Ritchey, Colorado Springs, Colorado
3:20- 4:00 p.m.	A Consideration of the Method of Attachment of Calculus to the Cementum. —Dr. Helmut A. Zander, Minneapolis, Minn.
4:00- 5:00 p.m.	Symposium demonstrating the use of the gingival biopsy in clinical correlation with various oral conditions.

The Histopathology Study Club of

The Colorado Dental Foundation

1. The Normal Oral Mucous Membrane . . . . . B. Orban
2. Gingivosis and Lichen Planus . . . . . Cal L. Foss
3. Herpetic Stomatitis and Pemphigus . . . . . H. E. Grupe
4. Hyperkeratosis and Leukoplakia . . . . . John M. Nabers
5. Psoriasis and Lupus Erythematosis . . . . . A. A. Ariando
6. Disturbances in Pigmentation and Fordyce Spots . . . . . Charles W. Finley

Saturday—September 6

9:00-11:00 a.m. Round Table Discussion of Traumatic Occlusion in Periodontal Disease.

Moderator, Dr. Wendell Wylie

Dr. Henry M. Goldman	Dr. Balint Orban
Dr. Bernard Jankelson	Dr. Clyde H. Schuyler
Dr. Arne G. Lauritzen	Dr. Lewis Fox

11:00-12:00 a.m. Business Meeting.

COMMITTEES

Program Chairman . . . . . Dr. Harold G. Ray
Clinic Committee Chairman . . . . . Dr. James Westbrook
Local Arrangements Chairman . . . . . Dr. Edward F. Allen
Press Chairman . . . . . Dr. Francis Fabrizio

CEMENTUM CURETTAGE

(Continued from page 177)

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